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THE SURGICAL TREATMENT OF HÆMORRHAGE ASSOCIATED WITH GASTRO-DUODENAL ULCERATION.[1]

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THE first result of a gastric or duodenal hæmorrhage will be a reduction in the volume of the blood within the vascular system. The average amount of blood in man has been estimated as being approximately one-thirteenth of the body weight; in other words, the volume of blood for a man weighing 64 kilograms (10 stone) will be somewhere in the vicinity of five and a half litres (10 pints). When a hæmorrhage occurs, its effect will depend not only on the amount of blood lost, but also upon the rate of occurrence of such exsanguination. In general, a sudden hæmorrhage will be more serious than a larger one spread over a more prolonged period, for the simple reason that a considerable interval must elapse before the maximum effect of the various compensatory phenomena can become effective.

Morbid Anatomy.

Although an acute peptic ulcer is not an uncommon cause of gastric hæmorrhage, this type of ulceration, on account of its multiple and disseminate characteristics, must perforce be relegated to the domain of the physician. With chronic peptic ulceration, on the other hand, the source of bleeding is almost invariably an erosion of an artery or vein lying in the base of an ulcer. Recent figures indicate that, although a history of hæmorrhage may be obtainable from about 20% of the ulcer patients admitted into our institutions, only about 10% of all peptic ulcers bleed.

In the case of the stomach the ulcer is usually situated either along the lesser curvature or on the posterior wall; when the duodenal bulb is involved, the ulcer, in cases of severe exsanguination, is practically restricted to the posterior wall. The mechanism is very similar to the bleeding that occurs in association with an infected wound; the artery wall is eroded and the lumen is opened up; hence spontaneous hæmostasis is uncertain. In point of fact, the hæmorrhage is correlated with an activity of the ulcerative process, and the patient not uncommonly states that the pain has been aggravated during the few days immediately preceding the hæmorrhage. Furthermore, it is very unusual for the pain to recur for several weeks after a hæmorrhage.

Morbid Physiology.

Irrespective of the site of a hæmorrhage, the immediate fall in blood pressure will be dependent upon a lowered blood volume, which necessarily diminishes not only the venous return, but also the output of the heart. The lowered blood pressure in turn leads to stimulation of pressor afferents in both the cardio-aortic area and the *sinus caroticus*, and the vasomotor centre thereupon endeavours to maintain unaltered the normal arterial pressure and incidentally the circulation through the brain as well. In other words, there is a reflex excitation of the sympathetic system, including an outpouring of adrenaline into the blood stream, which causes considerable vaso-constriction, thereby diminishing the total capacity of the circulatory system and tending to keep the blood as much as possible on the arterial side. By such a mechanism the capacity of the vascular system is greatly reduced and post-hæmorrhagic shock is warded off.

But, as Rous and Gilding have pointed out, this compensatory vaso-constriction is not exerted equally or indiscriminately throughout the body. It is greatest in

[1] A post-graduate lecture delivered at the Prince Henry Hospital on May 12, 1940.

the skin, and next in order come the spleen, muscles, glands and mucous membranes; the alimentary canal and other organs normally concerned with absorption of fluid are less affected. If widespread splanchnic constriction occurred, absorption would be interfered with and the replacement of lost blood would be extremely difficult.

It therefore follows that a small hæmorrhage (for example, up to one-tenth of the total blood volume) will have little or no effect on the arterial blood pressure; but a moderate hæmorrhage, whilst causing some reduction in blood pressure, will be accompanied by pronounced concomitant phenomena, such as a fall of venous pressure, increased respiratory movements, blanching of the skin, an increase in the blood sugar content, and a diminished flow of urine. At this stage the blood volume is still reduced and the percentage of hæmoglobin is relatively high. Absorption of fluid from the tissues gradually leads to restoration of the blood volume; *pari passu*, the concentration of the blood, the number of erythrocytes per cubic millimetre, and the percentage of hæmoglobin are reduced. In other words, when a gastric hæmorrhage occurs, the blood loss is replaced by tissue fluids, and the rapidity of this replacement will depend upon the ratio of the volume of available tissue fluid to the volume of blood lost. Small amounts, such as 350 to 700 cubic centimetres, are rapidly replaced; but, if amounts in excess of one-half of the total blood volume are removed, adequate replacement cannot occur for many hours. During this period every organ of the body will be inadequately supplied with oxygen, with consequent impairment of its functional efficiency.

On the other hand, the solid constituents of the blood are but slowly restored; the specific plasma proteins are obtained indirectly from the food and the blood corpuscles by an increased activity of the blood-forming cells of the marrow. From the analogy of animal experiments it may be argued that, if half to two-thirds of the hæmoglobin has been lost, 2% of the total hæmoglobin and 5% of the lost hæmoglobin will be restored daily, final recovery being thus delayed for three to four weeks. As the effective stimulus consists of oxygen lack due to diminished oxygen-carrying capacity, it should be remembered that if oxygen lack is severe (as after a major hæmatemesis), the red bone marrow may be so adversely affected that the process of repair will be considerably delayed. Furthermore, as the majority of fatalities do not occur for some considerable time (on an average six to seven days) after the onset of the hæmorrhage, it would appear that the general malnutrition of the tissues plays a much more important part in contributing to the fatal issue than does the actual loss of blood. In this regard it is interesting to note that autopsy revealed that the hæmorrhage had ceased (prior to the time of death) in 18 of the 39 fatal cases recorded at Saint Bartholomew's Hospital (1931 to 1935) and that in many of these cases bleeding had not occurred for several days prior to the fatal issue.

Estimation of the Severity of a Gastric Hæmorrhage.

It was formerly the custom to make repeated examinations (every two to four hours) of both blood pressure and hæmoglobin percentage in order that the degree of hæmorrhage might be estimated and any recurrence noted.

Blood Pressure.

Owing to the reduced blood volume and the lag in vaso-constrictor response, a sharp fall in systolic blood pressure occurs both during and immediately subsequent to a major gastric hæmorrhage. If the hæmorrhage ceases, a coincident fall in the hæmoglobin percentage, due to replacement of the blood loss by tissue fluids, will accompany a progressively rising blood pressure. In point of fact, the pressure gradually rises to within 15 to 35 millimetres of mercury of its customary level. It is therefore to be regretted that in many of these cases the normal blood pressure of the patient must necessarily be unascertainable.

In a prolonged major hæmorrhage the blood pressure is customarily maintained around about 100 millimetres of mercury until the various reserve mechanisms fail, when death ensues unless therapeutic aid is rapidly forthcoming. In other words, if the blood pressure falls below 80 millimetres of mercury for any considerable time, the oxygen requirements of the tissues will be unsatisfactorily met and secondary shock will supervene.

To sum up: although a general idea of the severity of an initial hæmorrhage and the moment of its cessation may be obtainable from repeated examinations of the blood pressure, this method of examination will form a very unreliable guide wherewith to estimate the extent of blood loss.

Hæmoglobin Value.

A reliable estimation of the total blood loss cannot be computed from the hæmoglobin percentage until such time as the volume of the blood has been fully restored; for example, a sudden loss of half the total blood volume would cause no immediate fall in the hæmoglobin percentage. Nevertheless, two-hourly measurements of both blood pressure and hæmoglobin percentage should be taken as a routine in all cases of gastric hæmorrhage.

Pulse Rate.

The pulse is rapid in every phase of hæmorrhage, being full and soft in the early stages; later, when adjustments occur, the tension improves, but the rate usually remains between 120 and 140 beats per minute. By a recurrence of the hæmorrhage the rate is further augmented; hence, in this respect, the pulse may be a more reliable index than blood pressure readings.

Blood Volume.

More recently the estimation of both blood volume and plasma volume has come into vogue and both have been appraised, in the early stages of a gastro-duodenal hæmorrhage, as being of greater value than hæmoglobin readings; as stated previously, a sudden loss of half the total blood volume would cause no immediate fall in the hæmoglobin percentage, whereas a blood volume estimation would at once register the extent of the loss.

An estimation of the blood volume is made as follows:

About 10 cubic centimetres of a 2% solution of Congo red in distilled water are slowly injected intravenously, and after five minutes a specimen of blood is withdrawn, oxalated and centrifuged. The concentration of the dye in the plasma is now compared colorimetrically with a known dilution of the dye injected, and from this the plasma volume may be calculated.

Portion of the aspirated blood is drawn into a hæmatocrit tube and centrifuged for twenty minutes in order to determine the relative volume of corpuscles and plasma.

If R is the percentage reading of test solution, $\frac{200}{R} \times 10$ cubic centimetres of dye injected $\times$ 100 = the amount of plasma in cubic centimetres.

The blood volume is calculated from the hæmatocrit reading.

$$\text{The total blood volume} = \frac{100 \times \text{amount of plasma (c.c.)}}{\text{Percentage of plasma in blood.}}$$

This estimation can be very easily carried out during the progress of a continuous intravenous transfusion and will accurately indicate whether the hæmorrhage or the transfusion is gaining ground.

Blood Urea Content.

The anoxæmia and impaired circulation consequent upon a major gastric hæmorrhage usually lead to temporary impairment of the kidney function, and a pronounced rise in the blood urea level is the result. Figures between 75 and 150 milligrammes per 100 cubic centimetres of blood are commonly recorded on the day following a severe hæmorrhage, and a sudden increase in the level would indicate a recurrence of the hæmorrhage. A reading of between 300 and 400 milligrammes per 100 cubic centimetres of blood is not unusual shortly before death.

Exclusion of Other Disease Conditions.

Apart from gastro-duodenal ulceration, whether simple or malignant, a large number of pathological states may give rise to hæmatemesis or melena as a symptom, and such conditions must be carefully excluded. In diseases associated with splenomegaly or with hepatic cirrhosis or

obstruction *et cetera*, surgical interference is either obviously contraindicated or, if demanded, certainly involves no direct attack on the stomach itself.

Degree of Exsanguination.

In order to assess the value of any given treatment it becomes essential that cases of gastro-duodenal hæmorrhage should be graded according to their severity.

A useful classification is into the following three groups:

I. *Moderate Exsanguination.* The hæmoglobin value falls to between 65% and 50%, or up to one-third of the cell volume is lost. Slow but progressive recovery usually ensues and the total mortality rate should not exceed 5%.

II. *Severe Exsanguination.* The hæmoglobin percentage lies between 50 and 35, and approximately 15% of the patients die as a result of the hæmorrhage. Severe impairment of bodily function will be present; but after a period of compensatory adjustment there is a tendency to spontaneous recovery, provided no further hæmorrhage ensues.

III. *Critical Exsanguination.* In this group the hæmoglobin percentage is reduced to below 35, or over one-half of the cell volume is lost, and the mortality rate is approximately 40%. The power of readjustment usually fails, and death will ensue unless restorative measures are rapidly forthcoming.

Medical Treatment.

Every medical practitioner may at any moment be called upon to treat a patient suffering from gastro-duodenal hæmorrhage, and there are few conditions in which life or death depends so directly upon the treatment adopted. As the mortality rate throughout the world in cases of gross hæmorrhage varies from 15% to 30% (Blackford), it is advisable to transfer the patient at the outset to a well-equipped and fully organized hospital.

The orthodox medical treatment in cases of moderate exsanguination includes the following: meticulous care for at least a week; the absolute withholding of anything by mouth for thirty-six hours; morphine in sufficient doses to produce twilight sleep for the same period; and most careful nursing both by night and by day. During the period of starvation the patient's mouth should be kept scrupulously clean.

At the first opportunity the patient's blood should be typed and half a gallon of appropriate blood should be available should necessity arise. The customary transfusion of one pint (568 cubic centimetres) of blood is woefully inadequate. After all, for a man weighing ten stone, one pint of blood is only one-tenth of the normal blood volume and could not be expected to raise the hæmoglobin percentage by more than 10.

In the preliminary stages tap water should be given *per rectum*, 15 ounces being run in every four hours, or preferably the continuous drip method may be employed. F. A. Coller estimates that a sick patient requires at least six pints (three and a half litres) of fluid per day. Nothing like this quantity is ever given to a patient with gastro-duodenal hæmorrhage when he is treated by orthodox measures. Hence water should be regarded as being second only to oxygen in importance, and in the treatment of such patients an adequate intake of fluid must take precedence over even "a liberal ulcer diet". Further, there is no evidence available to prove that the administration of fluid or pultaceous food by mouth stimulates further bleeding.

A few years ago Professor E. Meulengracht, of the Bispebjerg Hospital, Copenhagen, published a large series of cases with a death rate of only 1%, in which liberal feeding from the outset, together with the administration of alkalis, had replaced the customary régime. Confirmation of Meulengracht's claims in a large series of cases is urgently awaited. Between thirty and forty years ago Lenhartz introduced a similar method of immediate feeding, but after a few years this procedure fell into disuse.

R. V. Christie and G. Lovell Gulland have shown that anything less than 10% of hæmostatic serum has no appreciable effect *in vitro* on the coagulability of hæmophiliac blood. Hence, as four to five cubic centimetres added to the six litres of circulating blood can only produce a maximum concentration of 0·1%, it is obvious that such doses can have little effect on coagulability.

With critical exsanguinability the main aim should be to restore the blood volume and hæmoglobin percentage to their normal figure as rapidly as possible. Prolonged anoxæmia only causes irreparable damage to vitally important cells; for example, immediately after a critical hæmorrhage the patient is usually quite rational, whereas some three or four hours later he becomes drowsy and stuporose. Further, if the patient is kept in that state, the hæmorrhage tends to recur, because the clot appears to be organized very slowly in such cases.

Dextrose and Physiological Saline Solutions.

Owing to the rapidity with which they are diffused through the capillary walls, dextrose and sodium chloride are much less effective than blood in controlling post-hæmorrhagic shock. If given in excess they contribute to the widespread œdema so commonly seen, especially in the lungs, in fatal cases of gastric hæmorrhage.

Citrated Blood.

The researches of Cullinan and Price have clearly demonstrated that the intravenously administered "blood drip" does not act as a hæmostatic. Although a transfusion[1] may augment a hæmorrhage if given while bleeding is still going on, there is no evidence that such a procedure can initiate a recurrence when once the hæmorrhage has ceased. Hence a continuous intravenous drip transfusion should not be used as a routine measure, but only as a remedy for post-hæmorrhagic shock. It must, however, be employed whenever the systolic blood pressure falls below 90 millimetres of mercury, if the hæmoglobin level falls below 40%, if the pulse rate rises above 130 beats per minute, or if the blood urea level rises above 100 milligrammes per 100 cubic centimetres of blood.

With such treatment the mental confusion rapidly disappears, the blood pressure returns to some 20 millimetres below its normal level, the hæmoglobin percentage reaches somewhere in the vicinity of 80, and the blood urea level falls; but the pulse rate, although the volume greatly improves, usually remains in the vicinity of 90 to 100 beats per minute for several days. The blood is usually allowed to run in at the rate of five ounces (roughly 40 drops per minute) per hour, and the administration is continued for one to three days. A selective retention of the corpuscles without any increase in the plasma is found to occur, and the hæmoglobin should be augmented by 10% (one pint of blood) every four hours.

Computation of the Amount of a Blood Transfusion.

It has been estimated that 15 cubic centimetres of blood per kilogram of body weight will raise the blood count by one million red cells. Thus:

1 kg. = 2·2 lbs. = 15 c.c. blood = 1,000,000 red blood cells.

Hence a man weighing 10 stone should be given

10 × 14 ÷ 2·2 = 64 kg. × 15 c.c. = 960 c.c.; that is, 34 ounces.

Surgical Treatment.

As stated previously, surgical intervention is contraindicated in acute peptic ulcer, because bleeding from such a source may be more efficiently controlled by medical treatment, combined, if necessary, with a transfusion of blood. Further, there will be no external manifestation of the presence of the ulcer, and the stomach must be opened in order to locate the bleeding point or points.

Hence the main indications for surgical interference will be those cases of chronic peptic ulcer in which the bleeding persists or recurs in spite of medical measures, more especially if the patient is over fifty years of age and has a long history of gastric disorder. W. E. Chiesman, of Saint Thomas's Hospital, has pointed out that when the hæmorrhage continued or when it recurred within twenty-four hours of the commencement of medical treatment, the mortality rate was in the vicinity of 74%, whereas if five to six days had elapsed without recurrence

[1] Christiansen (1934) states that the mortality rate has doubled in the Kommune hospitalet, Copenhagen, since intermittent transfusion (phleboclysis) had become a common practice.

the prognosis was usually good. However, no operative treatment should be contemplated until the hæmoglobin percentage has been restored to 65 or over; hence the employment of the continuous intravenous drip blood transfusion before, during and subsequent to the operation must be looked upon as an essential adjuvant to a surgical incursion. By means of such pre-operative transfusion a moribund patient may be withdrawn from the danger list in less than thirty-six hours and converted into a moderate surgical risk within two or three days.

In the past the surgical treatment of gastro-duodenal hæmorrhage has had a "bad Press" because operations were performed too late and frequently were unsuitable. Victor Pauchet considers that it is not possible to secure hæmostasis by operations such as gastro-jejunostomy, jejunostomy, ligature of vessels at a distance and sequestration or infolding of the ulcer; but patients sometimes recover in spite of, and not because of, these operative procedures. In other words, indirect measures are unlikely to control bleeding, and they generally prove futile; hence the surgeon must make a direct attack at the very source of the bleeding. This is best done by securing the actual bleeding point, by some form of local excision, or even by partial gastrectomy.

In practice the actual operative procedure will in great measure be determined by the state of the patient, the condition of the ulcerated area and the skill of the surgeon. Ligature of the bleeding vessel or vessels through a gastrotomy or duodenotomy opening will almost certainly arrest the bleeding; but such a procedure does not involve any attack on the ulcer *per se*. A useful adjunct to the above operation will be ligature in continuity of the gastro-duodenal artery; but œdema and infiltration around a callous duodenal ulcer may render such a procedure extremely difficult.

Other surgeons favour local resection of the ulcer, either by knife or cautery (which may or may not be combined with posterior gastro-jejunostomy) or even partial gastrectomy.

If the patient's condition is satisfactory and the parts lend themselves to excision, a partial gastrectomy of the retrocolic Pólya type should be undertaken, for such a procedure is the only one likely to be followed by permanently satisfactory results.

In the Mayo Clinic series Eusterman and Balfour state that only in 22% of cases of hæmorrhagic duodenal ulcer was a resection operation possible. When local conditions prohibit partial gastrectomy, Professor Hans Finsterer's operation has been suggested; but such a manœuvre does not involve any direct attack on the ulcer. However, after transection of the stomach local treatment may be easily and simply effected by removing the lower clamp and widely opening up the anterior wall of the distal segment so as to obtain an unobstructed view of the ulcer. Hæmorrhage having been controlled, the greater part of the pyloric portion is then freed and excised; but a sufficiency of the prepyloric area must be retained to secure easy and efficient closure.

Conclusion.

The day will ultimately come when medical science will be able to prevent peptic ulceration; but until such a Utopia arrives gastro-duodenal hæmorrhages will continue to occur and to recur, seriously threatening life and often ending fatally. At the present time the only inevitably fatal cases are the extremely rare ones of "*hémorragie foudroyante*", in which death occurs during the first hæmorrhage. In all other cases it should be possible to tide the patient over sufficiently long for it to be possible to deal directly with the ulcer by operation.

In conclusion, let me reiterate the statement that a gastro-duodenal hæmorrhage may occur in a variety of circumstances having an entirely different therapeutic significance. The bleeding may be small and without much risk to life, or massive and critical; the victim may be a young person or one past the prime of life; and finally there are first hæmorrhages and recurrent hæmorrhages. Such being the case, the therapeutic approach to each case must be considered individually.

THE TREATMENT OF PERITONITIS.[1]

By ALAN E. LEE, M.D. (Melbourne), F.R.C.S. (England),
Brisbane.

WHEN we were very young, and even at times much less remote, that popular diversion of the social tea-table, the clinical reminiscence, ended often with these foreboding words: "But peritonitis set in." Clearly, to the lay mind this signified a prelude to death and occasioned additional tragedy, in that it occurred chiefly in the illnesses of active adult life—appendicitis, peptic ulceration and the hazards of the child-bearing years.

I hope to show you that this reputation of former years, justified as it was by mortality statistics, is disappearing, and moreover, that this improvement is the result of the almost complete reversal of methods of treatment formerly in vogue.

Drainage, purgatives, the Fowler position, such *clichés* as "if the bowels can be made to act, the patient recovers" and "when in doubt, drain"—all are condemned by the inexorable facts of clinical experience. In September, 1939, at the Queen's Hospital, Honolulu, a death from perforated gastric ulcer was discussed at the weekly staff meeting. Many of America's leading surgeons, gathered in Hawaii for the Pan-Pacific Surgical Congress, were asked individually by Dr. Larsen, the medical director, what they did with regard to drainage. To such an extent had the pendulum swung that not one surgeon would admit that he ever drained the abdominal cavity in cases of peritonitis. Such a reversal of opinion has occurred, first, because of better knowledge of the dangerous factors in peritonitis, and secondly, because of better methods of controlling these factors.

Before I discuss treatment a few words are necessary on other aspects of this disease. In the vast majority of cases peritonitis is caused, in order of frequency, by diseases of the appendix, the pelvic organs, the gall-bladder and the stomach and duodenum, or follows operations on the colon and rectum. Clinically we can distinguish three varieties: local, spreading and diffuse peritonitis.

Cases of local peritonitis comprise those in which an early spread beyond the original focus has been limited by time or walled off by the defensive mechanisms of the abdomen. The symptoms are those of the original lesion with superimposed local pain and tenderness and muscle spasm.

Spreading peritonitis is applied in those cases in which the peritonitis begins locally but continues to spread over a wide extent of peritoneum, no walling-off process taking place because of the virulence of the infecting organisms or because of ill-advised treatment. The process may be eventually walled off at a distance from the original focus, or the infection may continue until death ensues. The prognosis in such cases depends on the amount of preparatory reaction the peritoneum has made before the infection began to spread, on the nature of the organism, and on whether the original infection continues to pour into the cavity.

Diffuse peritonitis may be a terminal picture of the spreading type; but it may also be initiated by the sudden outpouring of infective material from the perforation of a viscus occurring so suddenly that the peritoneum does not experience a preparatory reaction; in other words, an open rupture occurs. Wide areas of the peritoneum are affected at the same time. The onset is sudden and fulminating, the pain is excruciating and the constitutional reaction is severe.

The pathological process in peritonitis spreads from the focus of infection. The pink injected membrane becomes coated with a layer of fibrin; this makes it sticky and causes coils of bowel to be gummed together. A fluid exudate collects, and as the disease advances both the fibrin and the exudate become increasingly purulent. Coils of small intestine lying in these pools of pus become inflamed and paralysed, and the resulting obstruction may be complete. As the result of fibrinous adhesions, fixation

[1] Read at a meeting of the Queensland Branch of the British Medical Association on April 5, 1940.

and distension of gut may produce kinking with mechanical obstruction. The regression of the generalized phase may leave areas of walled-off inflammation—subphrenic or abdominal abscesses.

In comparison with earlier years, the emphasis in peritonitis has moved away from the toxic effects of the peritoneal inflammation *per se*, and has become concentrated rather on its effects on bowel function. It is now quite well recognized that the fibrin and the exudate are part of a protective mechanism, no less in the generalized case than in those in which it succeeds in walling off an intraabdominal abscess by building up a local barrier. The removal of this generalized exudate may be as disastrous as the breaking down of an abscess wall. Treatment is good only when it aids the natural defences of the body and restores the normal physiological reactions of the patient.

The problem of peritonitis, then, is that of a constantly seeding focus, opposed by a defensive mechanism, the peritoneal reaction or peritonitis, benign in itself, but harmful in its secondary effects on the gut function. These secondary effects may be summarized as follows: (*a*) toxæmia associated with gut distension and interference with the blood supply to the bowel wall; (*b*) toxæmic factors associated with mechanical intestinal obstruction; (*c*) biochemical factors associated with derangement of intestinal function; (*d*) toxæmia from residual abscess formation — abdominal, subphrenic, empyemic or in the abdominal wall.

When one is faced with a case of peritonitis, the first decision must be whether an immediate operation should be performed. If appendicitis is taken as an example of the precursor of peritonitis, I believe the answer must be as follows: (i) If the inflammation is limited to the appendix, if no peritonitis is present, there is no reason why prompt removal should not cure the process and the mortality rate be insignificant. (ii) In appendicitis with perforation the process of peritoneal infection may or may not be limited by natural processes. If there is evidence of such walling, as evidenced by mass formation in the region of the appendix, operation as an emergency measure is superfluous and can be attended only by an increased mortality rate.

If there is no evidence of such limitation of the inflammation, operation will depend on the surgeon's beliefs with regard to (*a*) the removal of a continually infecting focus, (*b*) the value of surgical drainage for peritonitis, and (*c*) the harm associated with an operative procedure at this stage.

With regard to the removal of a continually infecting focus, the perforated appendix has some likeness to the perforated peptic ulcer. If it is a fact that a continued seeding occurs from such an appendix, it would appear as necessary to close the source by removal of the appendix as to close the perforation of a peptic ulcer. One can safely say in this regard that theoretically, and on the assumption that it is associated with no unfavourable factors, removal of the appendix in spreading peritonitis is desirable.

Concerning surgical drainage of the abdominal cavity in peritonitis, which in former years was regarded as an essential part of its treatment, there is developing almost a complete reversal of opinion. As stated earlier in this paper, the dictum "when in doubt, drain" has become so outmoded that few surgeons in America will now admit that they ever drain the abdominal cavity in peritonitis. This modern aversion to drainage rests on very solid experimental and clinical evidence. Droegmuller and Heilman (*Surgery, Gynecology and Obstetrics*, Volume LIII, 1931, at page 726) produced peritonitis in dogs by opening a loop of ileum about 40 centimetres long, leaving the mesentery intact, and restoring the continuity of the gut round the loop. This gave essentially the same pathological picture as is seen in human diffuse peritonitis. The experimental peritonitis was fatal in 90% of the cases, with an average duration of four days. If at the end of twenty-four hours the isolated loop of the bowel was removed to clear up the focus of infection and the abdomen was closed without drainage, 42% of the animals survived.

In a second series of 20 dogs, in addition to the removal of the isolated loop, two rubber drains were inserted, one towards the pelvis and one into the upper portion of the abdomen, crossing the soiled area. Of the 18 dogs which survived this operation, 17 died of suppurative general peritonitis and one of streptococcal pneumonia—a mortality rate of 100%. The use of intraabdominal drains had increased the mortality rate of peritonitis in these animals from 58% to 100%. Droegmuller and Heilman concluded that drains in spreading peritonitis were worse than useless.

Clinical results bear out this virtue of non-drainage. Rhodes, in San Francisco, by withholding drainage, reduced the mortality rate of gangrenous non-perforated appendicitis from 9% to 0·9%, and that of gangrenous perforated appendicitis from 10% to 2·4%. Pelvic abscess formation was reduced in non-perforated appendicitis from 9·3% to nil, and in perforated appendicitis cases from 15·9% to 7·9%. These clinical results emphasize strongly that in no circumstances whatsoever should the abdominal cavity be drained as a treatment for peritonitis.

Surgical intervention in spreading peritonitis should be limited to removal of the focus with closure of the peritoneal cavity, provided that no great harm seems likely to result from such measures. At the same time all experienced surgeons must recall cases in which great harm was done by just such measures. Nature is constantly striving, even in advanced peritonitis, to wall off the primary focus, and frequently succeeds. Even the perforated peptic ulcer is often found to be closed at late operation or at autopsy. There can never then exist an imperative necessity for surgical measures to close a seeding focus. Moreover, we must all recall misfortunes in which a resistance that had developed seemed to have been quite destroyed by surgical intervention at the wrong time; and when a patient is very ill even small abdominal incisions under local anæsthesia go far towards tipping the scales between life and death.

Rhodes stated in this regard that when a patient with a perforated peptic ulcer was very ill he had no hesitation in substituting continuous gastric suction for surgical closure and obtained thereby gratifying results.

I would conclude this portion of my subject by offering the opinion that in spreading peritonitis surgical measures should be limited to closure of the continually seeding focus, and that even this surgical interference should be limited to the earlier stages of the process, when the patient is in a relatively good general condition. Later, less harm will be done by abstention from active surgical measures and concentration on the measures next to be discussed.

The Effect of Inflammation on the Gut Wall.

Many of the fatalities in peritonitis are associated with the effects of inflammation on the bowel wall, manifesting itself by localized failure of bowel function. If the peritonitis comes under control the process will be self-limiting; but meanwhile death may occur (*a*) from the results of distension, (*b*) from the biochemical upset induced by vomiting and the loss of fluid and electrolytes.

Though distension following abdominal inflammation has always been regarded as ominous, its significance is often ill understood. There can be no doubt that in moderation the distended coils exercise a protective action in limiting the inflammatory spread. Beyond a certain degree, however, the distension of itself becomes harmful, in ways presently to be discussed. However silent the abdomen may be to auscultation, the ileus is rarely complete, usually being limited to loops bathed in purulent exudate. Stasis of itself causes but little toxic absorption from the bowel; but when distension becomes great, the blood supply to the gut wall becomes defective. In such anoxic gut toxins are produced which on absorption via the peritoneal cavity may produce lethal effects.

The control of distension is, then, a very important part of the treatment of peritonitis. In past years this was attempted by such methods as purgation, gut stimulants (pitressin, acetylcholine, eserine) and surgical drainage of the gut by ileostomy or jejunostomy. In more recent years

decompression by gastric or duodenal intubation with continuous or intermittent suction has been employed; but it is probable that in the future all these methods will be superseded by the use of the Miller-Abbott tube. Even in the most complete adynamic ileus a small balloon attached near the apex of the tube and inflated after being passed into the second part of the duodenum stimulates sufficient peristalsis to pass the tube down to the seat of obstruction, where by constant or intermittent suction all the obstructive and distensive factors can be controlled. At the same time, if the obstruction is sufficiently low, it allows of mouth feeding with possibilities of absorption in the gut above the obstruction. The tip of this tube is of metal, so that if the obstruction proves to be of mechanical origin, a flat X-ray film will reveal the exact site of the block (with reference to the abdominal wall) and so allow of its surgical undoing with a minimum of trauma.

What is the origin of the fluid that, even in the presence of complete abstention from mouth feeding, can be aspirated from the upper part of the gastro-intestinal tract in small-gut obstruction? The manner in which the vomitus is often spoken of as becoming gradually fæcal would suggest a belief in the mind of the speaker that the obstructed gut empties itself from above downwards. This is a very incomplete presentation of the truth.

The intestinal tract in this regard can be considered as having three segments with differing function. The upper few feet consist of a secretive area, into which are poured every day many pints of saliva, bile, gastric and pancreatic secretion and *succus entericus*. In the lower part of the ileum and right side of the colon there is an absorptive area where this fluid is mainly reabsorbed after it has carried out its digestive function. The left side of the colon is a reservoir where waste products can be stored till a convenient situation exists for evacuation.

If a block exists between the second and third segments, emptying of the bowel is still possible and the internal fluid circulation is intact. Apart from waste residue, the contents of the upper part of the intestinal tract will be absorbed adequately in the lower part. There may be distension, but there need not be vomiting.

If a block exists between the upper part of the intestinal tract and the absorptive area bowel action is still possible (even repeated bowel action). But because the circulation of the internal fluid is disrupted there will collect in the upper portion of the gastro-intestinal tract not only whatever fluid is ingested, but also this large volume (two pints or more daily) of gastro-intestinal secretions. There will be distension, and vomiting will occur unless this accumulation of fluid is cared for by some method of œsophageal intubation.

This simple explanation points a fundamental lesson, that obstruction in the small intestine is not demonstrated by an ability or inability to empty the colonic reservoir, but by the persistent accumulation in the upper portion of the gastro-intestinal tract of about two pints daily of fluid above the amount ingested. Failure to understand this has been the cause of the loss of many valuable lives.

As was stated earlier, obstruction due to inflammatory ileus is a self-limited disease, and return of function follows a day or two behind the subsidence of the temperature and the general signs of peritonitis. Such a return of function is heralded by a diminution in the volume of retained fluid and a lessening of the abdominal distension. Till such signs of returning function are manifest, any positive measures to empty the intestines are contraindicated. Such measures include purgatives, whose use alone has been responsible for converting thousands of simple cases of limited peritonitis to the spreading and diffuse forms. Enemata are likewise contraindicated; their repeated use wears out the patient and can at best only empty the colon. A good maxim is: Let the bowels alone, and as the body defences against the infection begin to gain the tone of the gut will return.

A problem to test the most expert surgeon is the decision whether continued distension following intraabdominal inflammation represents a mechanical obstruction. Quite frequently the earlier inflammatory ileus merges imperceptibly into the mechanical form. The text-book differentiations concerning colicky pain, the "silent abdomen" *et cetera*, are soon shown to be unreliable. The following points, however, merit great consideration. The fact of small intestinal obstruction (but not its mechanism) can be observed certainly by means of an indwelling gastric tube.

During the acute phase of peritonitis the path of wisdom is to regard the obstruction as inflammatory; and the same rule is safe perhaps for forty-eight hours after the temperature becomes normal. If the gastric signs still indicate a complete obstruction, it is imperative to regard it as mechanical and to act accordingly.

The peritonitic process is also distinctly limited in its time incidence. After an attack of perforative appendicitis a maximum of about one week may be allowed for the generalized stage of a peritonitic reaction to subside. Any later signs of obstruction, even though they are associated with residual localized inflammation, must be regarded as mechanical rather than inflammatory.

The Treatment of Mechanical Obstruction following Peritonitis.

The surgical measures necessary to relieve a mechanical obstruction should be the simplest available in the circumstances. Occasionally the drainage of an abdominal abscess will be all that is necessary to restore the lumen to a loop of small gut in its wall. To undo obstruction, the approach to the obstructed loop should be as direct as possible, and in this relation the evidence afforded by a flat X-ray photograph of the abdomen, and particularly by an X-ray film taken with the metal tip of the Miller-Abbott tube at the site of obstruction, should not be dispensed with.

Rather than conduct a difficult exploration amongst loops of distended gut, it has often seemed wise to turn these loops out through the abdominal wound. The loosening of the inflammatory attachment of the obstructed loop is usually not difficult; but in some late cases, in which several loops are firmly attached round an area of localized suppuration, it may be much simpler to by-pass these loops by a lateral anastomosis than to attempt their separation.

The Management of Biochemical Changes in the Blood.

Water, salt and nourishment are three substances essential to body metabolism. The instincts of hunger and thirst will provide for an adequate intake of these essentials in most individuals. But when, as in peritonitis, the oral administration of fluids is not tolerated and the surgeon must supply these body essentials parenterally, he is confronted with two problems: (i) What volume of fluids should be administered? (ii) What kinds of solutions should be used?

To answer these questions, one must first consider the normal water requirement of the patient. It is obvious that if the water balance of the body is to be maintained the water intake must equal the water output. Water is eliminated from the body in its process of carrying out two vital functions, namely: (i) the regulation of body temperature by the vaporization of water from the skin and lungs, and (ii) the excretion of waste products by the kidney. A small amount of water is also contained in the fæces.

The volume of water needed for the purpose of vaporization is larger than one might expect, ranging under normal conditions of temperature and humidity between 800 and 1,500 cubic centimetres of water. The process is little affected by the amount of water available, but utilizes what it needs to maintain body temperature even at the expense of the water content of the body. Its provision, in other words, forms an imperative need.

The amount of water in the urine varies directly with the water intake and indirectly with the amount of water otherwise eliminated. The absolute minimum water requirement for normal kidney functioning at maximum concentrating power is 500 cubic centimetres; but in order to ease the load on the kidneys and to provide a satisfactory excess, it is believed that sufficient water should be given to ensure a minimum urinary excretion of 1,500 cubic centimetres daily.

In addition, the abnormal water losses caused by such processes as profuse sweating, vomiting, gastro-duodenal drainage and drainage from wounds must be replaced volume for volume if the patient is to be kept in proper water balance. Patients suffering from fever may easily lose 2,000 cubic centimetres of fluid daily through their skin and lungs. Secretions amounting to about 8,000 cubic centimetres are poured into the upper part of the gastro-intestinal tract daily, and a considerable amount of this will be lost through vomiting or gastro-intestinal drainage. When possible, these abnormal losses should be carefully measured so that they may be accurately replaced.

The daily water needs of the sick surgical patient may, then, be summarized as follows (Table I):

TABLE I.

Water Needs.	Amount (Cubic Centimetres.)
For vaporization	800 to 2,000
For urine	1,500
For fæces	200
For abnormal losses	?
Total	2,500 to 3,700 (*plus* abnormal losses)

As a working rule, 3,000 cubic centimetres daily *plus* abnormal losses, will maintain a patient in a state of adequate hydration.

Dehydration.

The patient is often not seen at the beginning of his illness, but only after considerable dehydration has resulted. When the clinical signs of dry tongue, dry skin, sunken eyes, and scanty concentrated urine are present, experiments have shown that such a patient has lost fluids amounting to about 6% of his body weight. For the patient weighing 140 pounds this amounts to about 4,000 cubic centimetres. To restore the fluid balance this amount of fluid must be added to the quantities estimated above.

The kind of fluid that should be given depends on a consideration of the salt requirements of the body. In the light of our present knowledge we believe that the sick surgical patient should have a basal salt intake of four to five grammes daily. When a patient loses fluid abnormally from the body he invariably loses electrolytes. The sodium chloride content of gastro-intestinal secretions varies; but it is never greater than the salt content of physiological saline solution. It is a good working rule, then, to replace abnormal fluid losses volume for volume with physiological saline solution.

The dehydrated patient inevitably has also a low plasma chloride content. If the plasma chloride content is known, and also the patient's weight, the amount of salt necessary to raise the salt content of the body to the normal level can be calculated. This should be replaced as normal saline solution, intravenously, at the rate of about 500 cubic centimetres per hour.

It is now possible to state how the body fluids should be maintained.

Physiological saline solution should be used: (i) to supply the basal salt needs of the body (500 cubic centimetres per day); (ii) to replace abnormal fluid losses, volume for volume, or by calculation, if the amount lost is unknown. The rest of the water requirements of the body should be provided by a 5% solution of glucose in distilled water.

When both the water and the salt needs of surgical patients who cannot take fluids by mouth are considered, the daily parenteral fluid requirements of such patients may be summarized as follows:

A. A patient not losing fluid abnormally:
- (i) Basal salt needs (four to five grammes), 500 cubic centimetres of physiological saline solution.
- (ii) Water for vaporization and urine, 2,000 to 3,000 cubic centimetres of 5% glucose solution in distilled water.

B. A patient losing fluid abnormally:
- (i) Abnormal losses to be replaced volume for volume with physiological saline solution (this usually provides for the basal salt requirements as well as the abnormal salt losses).
- (ii) Water for vaporization and urine, 2,500 to 3,500 cubic centimetres of a 5% solution of glucose in distilled water.

General Measures for Increasing the Defence Mechanisms of the Body.

The application of an ice-bag to the abdominal wall has been a time-honoured remedy in treating peritonitis, and its local anæsthetic effect has done untold harm in delaying operation beyond the local stage. There is no evidence that it has any effect on the course of the disease. Heat is often grateful, but it is also doubtful whether it affects the course of the disease.

Posture was regarded at one time as important in localizing infection. Coller stated in this regard that its use was based on two misconceptions: first, that absorption was less rapid in the pelvis, and secondly, that with the trunk held at an angle of 45° drainage would be downward. Neither of these assumptions is true, and the best position in bed for the patient with peritonitis is that which is most comfortable to him. Often this may be the sitting posture, because with a distended abdomen breathing is then easier than in lying positions.

The use of inhalations of 100% oxygen is having a certain vogue in the United States of America at present. One of its virtues is thought to be the diminution of intestinal distension. The gaseous content of distended gut is said to have a high nitrogen concentration, and if the blood is freed of nitrogen by the continued breathing of pure oxygen, absorption of this gas occurs from the intestines, with relief of distension.

Sulphanilamide has provided a powerful remedy against the common organisms in peritonitis. There is no doubt at all that improvement frequently occurs when it is employed. American opinion holds that its use should be controlled by daily blood estimations and that during the whole acute phase of the disease a blood content of 11 milligrammes per 100 cubic centimetres should be maintained.

Protection against Peritonitis.

For a number of years many investigators have been endeavouring to develop a substance that would render the peritoneum immune to contamination at operation by bringing into play its defensive mechanism before contamination and infection occurred. Nature can do this gradually, as witness the apparent immunity to contamination of the peritoneum surrounding a colostomy or fistula. As a prophylactic before operations on the colon and rectum various substances have been used in North America. A vaccine of killed *Bacillus coli* and streptococci seemed to give excellent results at the Mayo Clinic; but one of its early users, Rankin, has now discontinued it.

At present "Coli Bactragen" is much used. It is a substance composed of killed colon bacilli in a solution of 1·5% gum tragacanth. It is supplied in collapsible tubes, and if contamination of the peritoneal cavity occurs at operation it can be introduced with the certainty that it will produce a protective reaction within three hours and so forestall any infection that may take place during the surgical procedure. This substance goes far towards stimulating the defence reaction that occurs naturally around a colostomy or a fistula.

I was recently told by Dr. Balfour, of the Mayo Clinic, about work being done at the Rockefeller Institute in New York concerning the problem of defence against peritonitis. As I understood it, a substance made from the anaerobic organisms in soil that are responsible for the sterilization of sewage has been prepared, which can render the peritoneum of animals quite immune to contamination. It is at present too toxic for use in human beings, but Dr. Balfour was confident that the future holds promise of weapons against infection much more dramatic and valuable even than the sulphanilamide group.

Residual Abscess Formation.

Rhodes has demonstrated that non-drainage in peritonitis has resulted in a very pronounced diminution in residual infection, from 9·3% to 0% in non-perforative appendicitis, and in perforative appendicitis from 15·9% to 7·5%. Drainage of the abdominal wall, together with very loose suturing, is the main protection against abdominal wall infection. The other common sites for pocketing (the pelvis, subphrenic space and pleural cavity) present their own physical signs, and if these are watched for, and particularly if an X-ray examination is made on suspicion of subphrenic abscess or empyema, they should be rarely missed.

I would stress particularly that the drainage of these residual abscesses is not a matter of emergency, but is to be undertaken only after very mature consideration. The general condition of the patient, the degree of walling-off of the abscess, its relation to the abdominal wall and the site for incision, all these must be correctly estimated if damage is to be averted.

Summary.

In summing up the treatment of peritonitis it may be said that if a continuing focus of infection can be removed without any serious upset to the patient, this should be done. At operation the general peritoneal cavity and its contained exudate should not be touched; the parietal peritoneum of the abdominal incision should be closed, but the remainder of the incision should be packed or drained to minimize wound infection.

Distension should be relieved by the use of the Abbott-Miller tube, by the application of radiant heat to the abdominal wall, and by the inhalation of 100% oxygen; the chemical constitution of the blood should be kept normal by proper parenteral injections; morphine should be given in adequate doses to maintain rest and relief from pain, and the sulphanilamide level of the blood should be maintained at 11 milligrammes per 100 cubic centimetres.

A close watch should be kept for the development of localized abscesses, which should be drained; and if the paralytic distension should merge into a mechanical obstruction, it should be undone with the least possible trauma.

Under such a plan of treatment the mortality rate of diffuse peritonitis is capable of reduction to a figure under 10%.

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PARKINSONISM: AN ACCOUNT OF THE DISORDER AND ITS TREATMENT, WITH SPECIAL REFERENCE TO HIGH ATROPINE DOSAGE THERAPY.

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There appears to be sufficient reason for hoping that some remedial process may ere long be discovered by which at least the progress of the disease may be stopped.

JAMES PARKINSON.

Definition.

THE term "Parkinsonism" is applied to a clinical syndrome in which there occurs a "disturbance of motor function characterized chiefly by slowing and enfeeblement of emotional and voluntary movement, muscular rigidity and tremor".[3] Parkinson, in his "Essay on the Shaking Palsy" (1817) defined it as a condition characterized by "involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported, with a propensity to bend the trunk forwards, and to pass from a walking to a running pace: the senses and intellect being uninjured".[19] In brief, its characteristics are rigidity, tremor, and flexion deformity. It is important to realize that the term "Parkinsonism" is applied not to a disease entity, but to a syndrome which may occur as the result of a number of different pathological conditions affecting the *corpus striatum* or *substantia nigra* or both.

Types of Parkinsonism.

Recognition of the particular type of Parkinsonism is usually possible on clinical grounds and is of importance from the viewpoint of treatment.

Paralysis agitans was the original type, described by Parkinson as "the shaking palsy". It is characterized pathologically by a degeneration of the *corpus striatum* (senile or arteriosclerotic or both), whilst clinically it begins usually in patients over the age of fifty years, and its earliest manifestation is as a rule tremor.

Post-encephalitic Parkinsonism, on the other hand, is a disease which first appeared in 1917, when it occurred as a sequel to the lethargic encephalitis epidemic of that year. Since then it has become widespread. It was first adequately described by Buzzard in 1919.[5] Cases have since been continually occurring as sequelæ to the striatal damage wrought by subsequent epidemics of encephalitis. Characteristic features are its age incidence (in most cases it appears before the age of forty years), the preponderance of rigidity (which appears as a rule before the tremor), and a past history of some illness which can be interpreted as encephalitis. Post-encephalitic Parkinsonism is one of the commonest forms of the syndrome, and its incidence in early life makes it a most distressing and disabling malady.

Parkinsonism may occur as a result of cerebral arteriosclerosis leading to ischæmic degeneration of the *corpus striatum*; it may result from the action of toxins, for example, in manganese and carbon monoxide poisoning; and, rarely, it may follow trauma or neurosyphilis.

Treatment of Parkinsonism.

It is proposed to limit discussion on the treatment of Parkinsonism to the two important forms, *paralysis agitans* and post-encephalitic Parkinsonism.

Treatment of Paralysis Agitans.

The treatment of *paralysis agitans* has been summarized in the phrase "hyoscine therapy". The main disability in *paralysis agitans*, as stated, is tremor, and the sedative effect of hyoscine on this tremor was first observed in 1895 by Erb. Subsequent experience has confirmed this observation, and hyoscine (prescribed as hyoscine hydrobromide, $^1/_{200}$ to $^1/_{100}$ grain three times a day) has been found a useful palliative in this disorder. Its sedative effect is ascribed to a "selective action on the lower motor centres",[7] which action produces relief in tremor. Other drugs used are discussed in the next section.

Treatment of Post-Encephalitic Parkinsonism.

The treatment of post-encephalitic Parkinsonism is most important, for post-encephalitic Parkinsonism is a disease which affects young persons and which, with the exception of accidents, does not necessarily shorten life. Any relief, therefore, that can be given is of considerable importance.

Hyoscine, having been of considerable value in the alleviation of the symptoms of *paralysis agitans*, was naturally tried in the treatment of post-encephalitic Parkinsonism, in view of the close resemblance between the two conditions. It was found, however, to have little effect on the post-encephalitic tremor, which is usually less prominent than that in *paralysis agitans*. It did exert a definitely favourable effect on the rigidity.

Comparable doses of the drug were used for both diseases; but later Hurst[12] tried the effect of larger doses of hyoscine, the resultant dryness of the mouth and paralysis of accommodation being combated with pilocarpine. Hurst recommended the administration of a mixture of hyoscine hydrobromide, $^1/_{200}$ grain, with pilocarpine nitrate, $^1/_{10}$ grain, three times a day, the dosage to

be increased until the optimum was found. He considered that the use of a heavier dosage considerably enhanced the therapeutic effect. Patients continually receiving hyoscine medication, however, become mentally dulled and depressed, and they express considerable relief when the administration of the drug is discontinued (see below).

In 1928 Carmichael and Green[9] showed that stramonium, which contains atropine, hyoscamine and hyoscine, was more effective than hyoscine alone. Patients receiving this treatment, moreover, showed both mental and physical improvement (compare earlier remarks). Tincture of stramonium was given in doses of 45 to 60 minims three times a day, and it was found that in combination with pilocarpine even greater doses could be given with more favourable effect—for example, at least 60 minims of the tincture with two to five grains of pilocarpine nitrate.[12]

Various other preparations of stramonium, belladonna and their contained alkaloids were employed in treatment. There was no unanimity of opinion as to which was preferable, the general view being that with the usual method of administration there was no great difference between the various preparations of each drug.[1]

In 1935 came the introduction into medicine of Bulgarian belladonna. This was first advocated by Ivan Raeff,[22] an obscure Bulgarian apothecary, as part of a complicated regimen of powders and pills. These were shown by di Mattei[23] to have no useful pharmacological effect; but the Bulgarian belladonna enjoyed a tremendous vogue in Italy, owing to its advocacy by Panegrossi.[13] He considered that extracts of plants of the belladonna group grown in Bulgaria had a therapeutic effect which was superior to that of any of the drugs then in use. In his view it led to greater improvement, and the effect was maintained for some days after cessation of administration. Panegrossi appeared to be supported in his opinions by Neuwahl and Fenwick in England,[17] who in two cases thought the improvement obtained with Bulgarian belladonna was greater and more permanent, whilst there were fewer complications. Careful analyses made since,[12][15][1][16] have been unable to confirm these findings, and Alcock and Carmichael[1] consider that there is no difference between the effects of Bulgarian and English belladonna.

High Atropine Dosage.

Atropine was first employed in the treatment of post-encephalitic Parkinsonism by Radovici and Nicolesco[20] in 1921. It was used by them to relieve the myoclonic and choreiform movements of the actue stage, and in some of the cases relief was obtained. Later, however, Radovici[21] found atropine of greater value in the chronic stages of the disease, and it has since been widely used in one form or another for this purpose. Radovici recommended the use of atropine sulphate, subcutaneously, intravenously or by mouth, in a dose not exceeding two milligrammes *per diem*. It was found that the muscular rigidity was in some cases greatly relieved and that relaxation of the facies of Parkinsonism was often obtained. "One cannot speak of a cure of the disease, but of a symptomatic treatment giving relief."[21]

Bremer,[4] in 1925, drew attention to what is an almost diagnostic feature:[14] the unusual tolerance of these patients to atropine. He showed that as much as 20 milligrammes per day could be given without ill effect. It was on these observations that Kleemann,[14] in 1929, introduced treatment known as high atropine dosage therapy. Kleemann commenced by giving these patients 0·5 milligramme ($^1/_{120}$ grain) of atropine per day in the form of a 0·5% solution of the sulphate, one drop of which contained 0·25 milligramme. The dose was then increased by 0·4 milligramme per day, given over three doses, until maximal improvement was attained. Kleemann found the average optimal dose to be three to seven milligrammes ($^1/_{30}$ to $^1/_{10}$ grain) per day. Occasionally one-third or even four-fifths of a grain could be given with very little risk, and omission of a dose produced no ill effects. Hall[11] managed his dosage with tablets coloured according to strength.

The method of administration which I have found most convenient is the following. Atropine sulphate is ordered in 1% solution by the prescription of four grains of the drug to an ounce of *Aqua Menthæ Piperitæ*. Each drop (minim) of the solution will then contain $^1/_{100}$ grain of atropine sulphate. It is therefore a simple matter at any stage of the treatment to know exactly how much atropine the patient is receiving. The initial dosage used is one minim three times a day, which is increased by a minim a day for as long as improvement is obtained and no ill effects result. I have found that the average optimal dose of atropine sulphate is approximately one-third of a grain *per diem*. The condition of one patient, however, became stabilized on half a grain per day, whilst another found his optimum dose to be one-fifth of a grain. It will be seen that the optimum dose appears to be much higher than that found by Kleemann.[14]

Ill effects with this method have been surprisingly few, considering the magnitude of the dose. The only upset complained of is due to paralysis of visual accommodation. Whilst this does not affect the patient's mobility to any extent, difficulty is experienced in reading. An endeavour has been made to cope with this by lessening the morning dose and reciprocally increasing the evening dose. Temporary relief can also be obtained by the ocular instillation of 0·5% eserine drops; but this has been found unnecessary.

Dryness of mucous membranes, especially of the mouth, has not been troublesome. Indeed, so conspicuous and distressing a symptom is the profuse salivation that its arrest brings considerable relief to the patient.

Mental confusion, delusions, drowsiness, gastro-intestinal and vesical disturbances[10] are uncommon, and hyperthermia has not been observed.

Oculogyric crises, generally speaking, have been less frequent and less severe. "Prominal" has been found of no value in either preventing or arresting these attacks.

The advantages of high atropine dosage therapy are several. The formula used and the method of administration are simplicity itself. The patients are no longer under the depressant effect of hyoscine, and refer to their transference to atropine therapy with considerable relief. They feel and look brighter, and their physical condition is improved. Their rigidity and tremor are diminished, and their most distressing symptom, salivation, is relieved.

Mention must be made of important adjuncts to treatment. These patients should be out of bed each day and lead as active a life as they can for as long as possible. Routine tasks they gladly do, whilst occupational therapy helps to make their life a more interesting and happy one. "Benzedrine sulphate" has been found of value, especially when symptoms of drowsiness and lassitude are pronounced.[1][18] Massage and movements are useful in lessening rigidity and in preventing the development of contractures. By these means the Parkinsonian, even if not cured of his disease, receives effective relief of his most distressing symptoms and is allowed to enjoy the fullest life which his damaged *corpus striatum* will permit.

Summary and Conclusions.

1. The causes and types of Parkinsonism are considered.

2. The methods of treatment of Parkinsonism are reviewed, special emphasis being laid on high atropine dosage therapy.

3. The results of treatment are discussed. It is believed that at present the best results, both mental and physical, in the treatment of the post-encephalitic form are obtained with high atropine dosage.

4. A method of administration is suggested and its effects are reviewed.

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(29) I. Raeff: Cited by G. Panegrossi, *loco citato*.
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Reviews.

THE PINEAL ORGAN.

A VOLUME of 528 pages on the pineal organ might be regarded as representing an addition to medical literature of some considerable magnitude.[1] Unfortunately some serious reservations have to be made.

This joint work by a well-known surgeon and an anatomist has evidently been a labour of love. It is an effort not only to describe the human pineal organ, but to discuss the origin of the pineal body in the light of comparative anatomy. The result of this latter aim is that more than half the book is devoted to a comparative study of the eyes of invertebrates and the lowest chordata, to geological evidence and to a discussion of the relation of median and lateral eyes. It is this section on which the authors seem to have foundered. Perhaps, however, this fact may not be regarded as of much consequence, except that in a volume printed so well and set up with many illustrations the reader must pass over so many pages before reaching what can obviously be relied upon as fact and useful information. And let it be made clear that we have only praise for the sections dealing with the human pineal organ.

The first half of the book must be regarded as not only most disappointing, but as indicative of the danger that is present when experts leave their own sphere and invade the vast field of literature concerning the lower animals. The evidence that the authors are not familiar with the discoveries of the last thirty years is clear. More surprising still is the fact that their glossary and other pages indicate both a startlingly poor knowledge of the invertebrates mentioned and carelessness in proof-reading. Two or three examples will suffice to demonstrate this:

1. "Cirripedia—an order of the Ostracoda including the barnacles." The Cirripedia are the barnacles, but the Ostracoda are small crustaceans of entirely different structure.

2. "Diptera—an order of the Insecta having a single pair of transparent wings, e.g. bugs, plant lice." This is truly surprising. The house fly and the blowfly are Diptera; the bugs are an entirely different group belonging to the Hemiptera, which also includes the plant lice or aphids.

3. On page 65 we are told that *Gammarus ornatus* is a fresh water shrimp. On page 487, in the glossary *Gammarus ornatus* has become a gad-fly belonging to the order Hymenoptera!

4. "Amia (Gr. *άμία*, a kind of tunny): a bony fish allied to the mackerels." Now *Amia* is one of the most unusual of living fishes. It does not even belong to the same subclass of fishes as the mackerel.

These are really inexcusable errors, and there are many more. The most serious criticism we have to offer, however, is that in descriptions of invertebrate eyes, illustrations are taken from old works long regarded as out of date. Thus in the section on the molluscs, the well-known eyes of *Pecten* receive a great deal of attention; but the discussion is based on the work of Patten, that so much criticized author of the 1880's. The elaborate researches made since Patten's date are never mentioned anywhere, and this applies more or less to all the invertebrate descriptions.

It seems rather unnecessary to have tied so much discussion and theory on the invertebrate and vertebrate eyes to a valuable description of the vertebrate pineal organ. It is quite reasonable to indicate the ancient nature of the pineal apparatus in the vertebrate series, and the comparative study of this structure is valuable. In later chapters the authors discuss the possible functions of the organ and the pathology of pineal tumours. Descriptions of operative technique and a study of clinical cases follow before a final chapter on general conclusions in which comparative morphology is again in the ascendant.

[1] "The Pineal Organ: The Comparative Anatomy of Median and Lateral Eyes, with Special Reference to the Origin of the Human Pineal Organ Considered from the Clinical and Surgical Standpoints", by R. J. Gladstone, M.D., F.R.C.S., F.R.S.E., D.P.H., and C. P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., F.Z.S., F.A.C.S., F.R.A.C.S.; 1940. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 542, with 324 illustrations. Price: 42s. net.

AN ATLAS OF SKIN DISEASES.

THE second edition of Semon's atlas contains the 103 illustrations provided in the first edition, together with 17 additional plates.

In illustrations of diseases of the skin it is particularly difficult to depict truly the various combinations of colour which provide the typical appearance of the very varied lesions that have to be portrayed.

One cannot expect with mechanical processes like colour photography and printing to reproduce exactly the actual colours; but we feel sure that everybody who views the reproductions in this atlas will think that, if he sees on an actual patient any of the diseases illustrated, he will be able to recognize them much more easily than if he had seen only a photograph in black and white.

Dr. Semon has achieved considerable success in overcoming difficulties, and, in addition, the texture of the skin is beautifully reproduced. In many of the illustrations the reproductions have a distinct stereoscopic appearance. One of the many instances of this is to be seen in plate 52, showing two feet the soles of which are affected with psoriasis.

The text supplied to describe the diseases illustrated and the treatment to be recommended is also excellent. It is remarkable how much information Dr. Semon manages to include in the concise summaries which he supplies.

This is a production which will be a very valuable possession for any general practitioner. It will enable him to attain a higher degree of accuracy in diagnosis of diseases of the skin and provide him with a lead to rational treatment of these diseases.

Semon's atlas is a wholly British production, and this might be held as alone making it worthy of support; but, as it is, the quality of the plates and text are such that it can depend solely upon them to induce the practitioner to use the atlas as an essential addition to his professional library.

[1] "An Atlas of the Commoner Skin Diseases, with 120 Plates Reproduced by Direct Colour Photography from the Living Subject", by H. C. G. Semon, M.A., D.M., F.R.C.P., photography under the direction of A. Moritz, B.A., M.B., B.C.; Second Edition; 1940. Bristol: John Wright and Sons Limited. Crown 4to, pp. 284. Price: 42s. net.

The Medical Journal of Australia

SATURDAY, AUGUST 24, 1940.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

RESEARCH AND PUBLICATION: A NEW BENEFACTION.

As long ago as 1932, in a discussion on research and publication, the statement was made in these pages that the publication of research had a claim on endowment funds. We stated that the results of research were of little use unless they could be published so that workers in different centres might use them; and stress was also laid on the fact that if some articles were published without illustrations their value would be depreciated. It has been said that everyone likes a picture-book, and most medical readers will agree that a suitably illustrated article in a medical journal is more attractive than one devoid of such embellishments. Apart from this, however, there are certain branches of medicine, such as pathology and radiology, in which the illustration is practically as important as the letterpress describing it. To an expert, indeed, the photomicrograph, the drawing or the skiagram may be more important, for he can see what the author would describe, and he may form his own conclusions. There is every justification for the statement that the value of a publication, either by curtailment of illustrations or by their elimination, is considerably diminished. Unfortunately the illustrating of an article is often expensive. To be of much use, photomicrographs and skiagrams must be reproduced on art paper and fine-screen blocks are needed; a special printing must also be undertaken, and these all cost money. Few medical journals are sufficiently well endowed to be able to meet the expense incurred by the publication of many illustrations; so that both in Australia and in England authors are called upon to contribute to the cost of illustrations when the sum exceeds a certain amount. This is a hardship to some authors, and especially to younger research workers, whose remuneration as a rule is not generous. There has long been a need for a special fund, which could be used to help impecunious authors to pay their share of the cost of illustrations, and happily a start in this direction has been made.

In November, 1939, Professor C. G. Lambie, of Sydney, Dr. C. H. Kellaway, of Melbourne, and Dr. E. Weston Hurst, of Adelaide, approached the Walter and Eliza Hall Trust in Sydney with a request that the trust would make available a sum of money to assist in the illustration of medical publications in Australia. They advanced some of the arguments already put forward in this journal, and added that the cost of supplementing the allowance made by certain journals for the illustration of articles was a serious drain upon research funds. They pointed out that, though in other countries funds were available for defraying the cost of illustrations (for example, the Carnegie Trust in Scotland), no such funds existed in Australia. They also stated that the National Health and Medical Research Council had been approached in the matter, but would provide no grant for the purpose. The scheme suggested to the Trust was as follows:

1. That a sum not exceeding £100 in one year, be made available by the Trust for the purpose of defraying the expenses of publishing illustrations in the three medical journals of Australia, namely, THE MEDICAL JOURNAL OF AUSTRALIA, *The Australian Journal of Experimental Biology and Medical Science* and *The Australian and New Zealand Journal of Surgery*. It should be understood that the fund would only be drawn upon if necessary to supplement the allowance already made by the journals in question for this purpose. In the case of a university or research institution themselves publishing a monograph embodying work too extensive for publication in a medical journal the Trustees might also consider granting some assistance.
2. That assessors be appointed by the Trust for the purpose of advising the Trust in regard to grants to individual applicants.
3. That all applications to the Trust should be made through the editors of the journals concerned and that each application be accompanied by a copy of the article together with a statement by the editor as to whether the article is acceptable for publication, the total cost of the illustrations and the amount which the journal is prepared to contribute.

A reply was received recently from the Trustees stating in effect that they were prepared to adopt the scheme and that they had earmarked from the 1940 income £100 towards the cost of illustrating articles in THE MEDICAL JOURNAL OF AUSTRALIA and *The Australian and New Zealand Journal of Surgery*. They regretted that *The Australian Journal of Experimental Biology and Medical Science*, as it was published in Adelaide, did not come within the scope of the Trust, which was confined to New South Wales, Victoria and Queensland. It has since been decided that the following procedure shall be adopted. Papers will be submitted in the first place to the editor of the journal concerned, together with an application for a grant. On acceptance of the paper the editor will make a recommendation to the Walter and Eliza Hall Trust in regard to any application for a grant. Should either the editor or the Walter and Eliza Hall Trustees be in doubt regarding an application, the matter will be referred to assessors appointed for the purpose.

The Walter and Eliza Hall Trust, which has already done so much for medical science by adding to its already generous benefactions, has shown imagination and an appreciation of values for which we have waited many years; and in choosing to further medical knowledge in this non-spectacular way the Trustees have earned the gratitude of authors and readers alike. In the past the

need for such a fund as this has often been felt, and with printing costs mounting in every direction, as they are at present, this fund will be doubly welcome to authors. On behalf of the medical profession in the Commonwealth we offer grateful thanks to the Walter and Eliza Hall Trustees. The Trustees realize that more money than they have been able to provide will probably be required, and they have expressed the hope that others may be stimulated to follow their example. Many of our readers will reecho that hope; but they should be remined that only exceptionally do benefactions so unusual appear unsought. Had Professor Lambie, Dr. Kellaway and Dr. Hurst not sought the help of the Walter and Eliza Hall Trustees, the Trustees would not have known of the need and nothing would have been done. Medical practitioners know what assistance is required and can make appeals to those able to give it—even, it should be remembered, among their own ranks. Everyone will hope that further funds will become available from which contributors to *The Australian Journal of Experimental Biology and Medical Science* will also be able to benefit.

Current Comment.

A NEW TREATMENT FOR RHEUMATOID ARTHRITIS AND OTHER DISEASES.

IN his thesis for the doctorate of the University of Liverpool A. J. Helfet has propounded a new conception of the function of the parathyreoid glands and has applied it in devising a novel form of treatment for generalized fibrocystic and allied bone diseases and for rheumatoid arthritis.[1] It has long been generally accepted that the primary function of the parathyreoid glands is to control the amount of calcium in the blood; based on this premise various explanations of the mechanism of calcification and decalcification have been made, but they cannot always be reconciled exactly with clinical and experimental observations. From time to time the suggestion has been made in medical writings that the parathyreoid glands may exert their primary effect on phosphorus metabolism, but the possibilities of this idea have never been explored.

Helfet's theory is that parathormone regulates the amount of inorganic phosphate in the blood; that it prevents this amount from increasing to a degree that would upset any metabolic process in which the phosphates are concerned; that an accumulation of phosphate in the blood is a stimulus to increased secretion of parathormone; that the mode of action of the parathormone is by stimulating the excretion of phosphate by the kidney and by mobilizing calcium ions from the stores of calcium carbonate in the skeleton to form calcium phosphate, which is inactive and can be kept in solution in the blood under the influence of parathormone in greater quantities than normal; and finally that if the parathyreoid glands are removed the body will be unable to deal adequately with the blood phosphate. The theory is based on a variety of clinical and experimental evidence, observed and culled from the literature, and the author shows that it fits in with the phenomena of the parathyreoprival state, of tetany and of physiological and pathological hyperparathyreoidism.

Helfet proceeds to classify the symptoms of hyperparathyreoidism, which are general, skeletal and renal. The general symptoms comprise hypotonia, lassitude, anorexia, some degree of apathy, a tendency to coldness and bluish discoloration of the extremities, and sallowness of the complexion. The skeletal symptoms include osteoporosis, cyst formation and bone pain and tenderness, while the renal symptoms arise from increased excretion of calcium and phosphorus, which, if extreme, may lead to polydipsia and polyuria or to calculus formation. Helfet next discusses the role of hyperparathyreoidism in a number of diseases. In generalized fibrocystic disease of bone the relationship with hyperparathyreoidism has long been known; indeed, it can now be said that any condition of decalcification of bone with or without cysts, but accompanied by hypercalcæmia, hypophosphatæmia and a drainage of calcium from the system is due to hyperparathyreoidism. Even in those cases in which the levels of calcium and phosphorus in the blood are within normal limits at the time of examination, a state of "negative calcium balance" may sometimes be demonstrated, while a sallow complexion, atonia and apathy may be reminiscent of the clinical picture in hyperparathyreoidism. Rheumatoid arthritis is another disease in which several symptoms recall the clinical picture of hyperparathyreoidism—there are often sallowness, atonia, anorexia, loss of weight, numbness and pallor of fingers and toes in cold weather, while a state of negative calcium balance has been reported in this disease, and also absence of phosphoric acid from the urine. Again, in Paget's disease hyperparathyreoidism has been suggested as a causal factor. Helfet suggests that in this disease there is retention of phosphate in the blood for some reason and that the parathyreoid glands respond to their full capacity, the degree to which they are able to do so controlling the degree of decalcification of the bones which occurs in the first stage of the malady; once the glands become unequal to their task a state of relative hypoparathyreoidism exists and the reverse changes occur, this being the second and permanent stage.

It would seem reasonable in the treatment of hyperparathyreoidism not due to primary adenoma to try to reduce the intake of phosphorus and thus lessen the stimulus to the over-production of parathormone. This may be attempted by the administration of a diet of low phosphorus content, such as that of I. A. Anderson and A. Lyall, abstracted in our issue of April 13, 1940. Alternatively, and better, some substance might be given which would combine with phosphate in the intestine to form an unabsorbable substance. Helfet has employed salts of aluminium for this purpose. It is of interest that the only reference he has found to the prior use of aluminium in diseases of bone is one well known to medical historians. In 1845 W. MacIntyre sent to H. Bence Jones the urine of a patient suffering from "*mollities ossium*". It was in this that Bence Jones discovered the proteose that has since been associated with his name. At his suggestion "alum was added to the tonics in use with the view of checking the exhausting excretion of animal matter". Helfet advises that aluminium acetate be administered and that the quantity given should be enough to have a "phosphate-combining power" equivalent to 150 to 220 milligrammes of calcium daily. His prescription is as follows:

℞

Liquoris Aluminii Acetatis	ʒi ʒii
Syrupi	ʒvi
Essentiæ Cerasi Optimæ	miv
Mel Depuratum	*ad* ʒiv

Fiat Mistura.

Signetur: ʒi *quater die sumenda post cibum.*

The patient should have at least one pint of milk a day while taking this mixture. Helfet has more recently used aluminium gluconate, which he states is less unpalatable, but he does not give a prescription for this.

Helfet has applied this form of treatment to five patients suffering from generalized fibrocystic disease of bone not due to primary parathyreoid adenoma, to eight patients suffering from Paget's disease and to fifteen suffering from rheumatoid arthritis; fourteen of the cases are described in detail. The effects have been very remarkable indeed and most encouraging. No sign of aluminium poisoning was noted even in patients treated for more than a year. In the patients with fibrocystic disease the weakened skeleton has commenced to recalcify. In those with Paget's disease bone pain has become less frequent and severe, and fatigability has been reduced. In those with

[1] *The British Journal of Surgery*, April, 1940.

rheumatoid arthritis there was in the majority great improvement in general health and in the mobility of joints within a few weeks; even young men whose spines were rigid from ankylosing spondylitis lost their pains and regained some movement in their joints.

The physiology of the parathyreoid glands is undoubtedly complex, and most medical men do not attempt to understand it; Helfet's brilliant exposition and application of it and the encouraging results that have accrued in a common disease, though not yet confirmed, make it of interest now to everyone.

THE RELATIVE MERITS OF SUTURE MATERIALS.

LISTER made surgery safe for the patient. In doing so he created conditions suitable for the employment of non-absorbable sutures. There is irony in the reflection that Lister himself condemned silk and is largely responsible for the disfavour and distrust with which the majority of surgeons still regard non-absorbable materials. Surgical conservatism is nowhere better exemplified than in its blind preference for catgut. For over fifty years, workers, of whom Kocher and Halsted were pioneers, have shown the many respects in which silk is superior to catgut. In an editorial in *The Australian and New Zealand Journal of Surgery* in January of last year, H. R. Dew discussed the healing of surgical wounds and stressed, among other things, the wisdom of using silk in clean cases. The merits of non-absorbable sutures have been convincingly demonstrated both experimentally and clinically. C. H. Lipton used silk and catgut respectively in closing two parallel series of laparotomies, and reported, in *The American Journal of Surgery* of August, 1939, that lower temperatures and fewer wound complications occurred with silk. A. B. Longacre reported in *Surgery, Gynecology and Obstetrics* of February, 1939, that he had followed 925 cases of hernia repair, and analysed *inter alia* the relation between the results and the suture materials used. He found that the incidence of wound infection and of recurrence was approximately four times as high with catgut as with silk. The doubtful efficacy of catgut as a wound splint during healing is a matter of common observation. If a carefully closed incision has to be reopened after four to five days, either in the theatre or at autopsy, the appearance of the catgut sutures is such as to inspire the reverse of confidence. Many of the knots are untied, and the catgut for the most part has assumed a gelatinous consistency. The disintegration of catgut is accompanied by considerable local reaction. The details can be demonstrated histologically, but the reaction is seen macroscopically where catgut is used as a skin suture. Although a fine strand may have been employed, a red inflammatory zone soon appears around each stitch hole. This is not encountered around the skin sutures in clean wounds closed with silk or horsehair. In an endeavour to clarify the position, W. H. Meade and Alton Ochsner[1] have carried out extensive investigations to establish the relative values of catgut, silk, linen and cotton as suture materials. Three main aspects were examined: tensile strength, bacteriology, and tissue reaction and wound healing. Size for size, dry catgut was found to be the strongest material, followed in order by silk, linen and cotton. These materials were buried in the tissues with aseptic precautions, and tested again after varying periods of from two to fourteen days. The catgut used had been chromicized. The cotton remained practically unchanged, but the other materials showed a drop in tensile strength which was greatest in the case of catgut. Further, this substance developed considerable inelastic stretch. Tissue reaction after varying periods was examined in a series of laboratory animals. Meade and Ochsner found that, owing to an attempt on the part of the tissues to digest the suture by proteolytic ferments derived from leucocytes, absorbable sutures produced essentially a wet reaction. The excessive collection of fluid and leucocytes delays the appearance of fibroblasts. Non-absorbable sutures produce a dry reaction with minimal exudation, and fibroblastic proliferation begins early (three days as against five with catgut). The authors stress the importance of the rule, first enunciated by Halsted, that non-absorbable sutures must be interrupted and not continuous. Departure from this rule leads to sinus formation and brings the material into disrepute. The dry reaction to silk and allied materials is exemplified by the fact that thyreoidectomy and mammectomy incisions can be closed without drainage and without the accumulation of serum. Contrary to popular belief, Meade and Ochsner considered non-absorbable sutures superior to catgut in infected wounds, and found that severely infected wounds sutured with cotton healed without sinus formation and without extrusion of sutures. There are other advantages of non-absorbable sutures. They are easily and satisfactorily sterilized by boiling. Knots are more secure, owing to the higher coefficient of friction. Finally, the non-absorbable materials are obtainable at a fraction of the cost of catgut. In the above lines strong arguments have been presented in favour of the more general use of silk and cotton, at least in clean cases. Such arguments, it is pointed out, have been urged for many years. To avoid having to face an accusation of mental inertia surgeons should at least give unbiased consideration to the virtues of non-absorbable sutures.

[1] *Surgery*, April, 1940.

BROMIDE POISONING.

THE frequency with which bromides are used in modern therapy and the popular belief that a skin rash is the commonest evidence of bromide intoxication give special significance to a discussion of this condition by L. E. Harris,[1] who points out that the symptoms are predominantly nervous and a rash is uncommon.

Evidence of intoxication occurs if the drug is taken for a long time or in large quantities. Accumulation in the body occurs because, while the total halogen content of the body remains constant, the chloride ion is excreted more readily than the bromide ion and so is replaced by it. Diminished fluid or chloride intake or renal disease increases the rate of this accumulation. The onset of symptoms is usually insidious. Often unusual restlessness and excitability are the first symptoms, and more often than not result in an increase in dosage of the drug. Then come a slurring of speech, a generalized tremor and ataxia, making walking difficult. Swallowing may be difficult and the mucous membranes dry. Auditory and visual hallucinations are not uncommon. The reflexes of all types are depressed. In severe cases coma and even death may result. The diagnosis is suggested by the presence of bromides in the urine, and is established by the finding of a raised level of bromide in the blood. A figure of 150 milligrammes per hundred cubic centimetres of blood has been set arbitrarily as the level above which symptoms of intoxication appear. Therefore if a patient being treated with bromides becomes gradually more excited and tremulous, the correct procedure is not to increase the dose of the medicine, but to estimate the level of blood bromide.

Treatment consists of the administration of sodium chloride by mouth, as much as will be tolerated being given in the diet and in capsules. The author gave 20 grains four times a day in this way. Excess fluids should be taken. In severe cases intravenous infusions of a physiological solution of sodium chloride should be given. The bromides are thus gradually replaced by the chlorides and excreted. Even though restlessness is severe, other sedatives are usually ineffective and are better omitted. Harris reports the history of a child, aged four years, who for mild nervousness received 60 grains of bromide daily for five months, and that of a child who for epilepsy was given 45 grains of bromide daily for four months. Both developed severe symptoms of intoxication, and the blood contained 353 and 480 milligrammes of bromide *per centum* respectively. Both children responded well to treatment and recovered completely.

[1] *American Journal of Diseases of Children*, April, 1940.

Abstracts from Medical Literature.

DERMATOLOGY.

Contact Dermatitis.

JAMES W. JORDON AND E. D. OSBORNE (*The Journal of the American Medical Association*, November 25, 1939) report four cases of contact dermatitis from opium derivatives, three of which occurred in nurses and the fourth in a woman engaged in the manufacture of morphine tablets. The three young student nurses manifested erythematous papulo-vesicular eruptions of the skin of the fingers and hands, which at first were thought to be ringworm affections. It was found, however, that relapses of the dermatitis followed persistently when morphine substances were handled, whereas in the case of the morphine tablet moulder, the eruption developed on the flexor surfaces of the forearms and later spread to the hands, the back of the neck and eyelids. The authors found that in all four cases a positive result followed the intradermal patch test for morphine, and complete resolution occurred upon withdrawal of the patient from contact with the drug. The authors state that all their attempts at desensitization in morphine dermatitis have proved a failure, as they have found in other cases of the contact type of dermatitis. They are of the opinion that opium and its compounds deserve more attention as eczematogenous agents and as a cause of the contact type of dermatitis, and that this group of drugs should be suspected as a cause of eczematous dermatitis of unknown ætiology in nurses, physicians and pharmaceutical workers.

Sulphonamides in Lupus Erythematosus.

H. W. BARBER (*The Lancet*, March 30, 1940) states that most persons suffering from *lupus erythematosus* suffer severe reactions when sulphonamides are administered to them even in small doses. The symptoms produced comprise fever, scarlatiniform eruptions, tender lymphoglandular swellings, focal reactions in and transitory erythema around the patches, and temporary exacerbation of fibrositis or arthritis if these conditions should be present. In most cases, if administration of the drug can be continued, the lupus improves or disappears, as do any coincident symptoms of fibrositis or arthritis.

The Sugar Metabolism in Acne Vulgaris and its Treatment by Insulin.

H. C. SEMON AND F. HERMANN (*British Journal of Dermatology and Syphilis*, April, 1940) have investigated blood sugar tolerance curves of eleven patients suffering from *acne vulgaris* on not less than two occasions. They were able to demonstrate a "lag" as compared with the normal curve, an observation which implies a delay in sugar assimilation. Of eight women examined, seven displayed this abnormality. Five of the younger women exhibited an exaggeration of the lag at the beginning of menstruation. The results suggested a trial of insulin. Five units of zinc-protamine insulin were injected once a day at first, and later eight to ten units were injected two or three times a week. Two men were also treated, five and eight units being given respectively twice a week. All patients, without local treatment of any kind, showed some improvement. Four had not relapsed five to eight months after the injections were stopped. For the time being the authors leave it an open question whether the disturbance of the sugar metabolism favours skin infection by increasing sugar in the tissues, thus promoting growth of pyogenic cocci, or whether there is additional relationship between the hormone abnormalities and the skin, the sebaceous adnexa in particular, which may be corrected by the administration of insulin.

Role of Vitamin C in Various Skin Diseases.

W. F. LEVER AND J. H. TALBOTT (*Archives of Dermatology and Syphilology*, April, 1940) have investigated concentrations of vitamin *C* in the blood of 68 apparently healthy persons and in the blood of 181 patients seen in the dermatological clinic. No significant correlation was noted in the second group between the concentration of vitamin *C* in the blood and the development of lesions in the skin. The level of vitamin *C* in the plasma of the 68 persons ranged from 0·10 to 1·46 milligrammes per 100 cubic centimetres. The average was 0·6 milligramme. The authors quote reports from the literature which claimed clinical improvement in the following skin diseases after administration of vitamin *C* when deficiency of that vitamin had been found: *lupus vulgaris, lupus erythematosus*, generalized exfoliative dermatitis, eczema and purpura. They investigated the concentration of vitamin *C* in 181 patients in the dermatological clinic to see whether their findings confirmed or refuted the presumption that a deficiency of vitamin *C* in the body is a contributing ætiological factor in the production of skin disease. The average blood level of vitamin *C* for the 181 patients was 0·36 milligramme per 100 cubic centimetres; in 55% the level was below 0·30 milligramme. Throughout the series the values of vitamin *C* were scattered; in some patients they were high, in others low. In patients with psoriasis concentrations varied between 0 and 1·20 milligrammes, in urticaria between 0 and 1·12 milligrammes, in atopic eczema between 0 and 1·13 milligrammes, and in *lupus erythematosus* between 0 and 1·32 milligrammes. There were three diseases which had values below 0·30 milligramme per 100 cubic centimetres, namely, purpura, generalized exfoliative dermatitis and pemphigus. A short dietary history was taken of 117 patients. A high intake of vitamin *C* was assumed if fresh fruit was consumed at least five times a week; a low intake if fruit was eaten occasionally. On this basis 40 patients showed a high, 30 a moderate and 47 a low intake of vitamin *C*. Eighteen patients with a low level of vitamin *C* in the blood were given daily by mouth, over a period of two to ten weeks, 200 milligrammes of this substance. The average level in the blood after the experiment was 0·97 milligramme per 100 cubic centimetres. Five patients with urticaria, four with *lupus vulgaris*, three with eczema, two with generalized exfoliative dermatitis, two with psoriasis, one with purpura and one with pemphigus were treated. None of the patients manifested improvement which could be attributed to the high intake of vitamin *C*.

Pruritus Ani and its Relationship to Seborrhœic Eczema and Dermatophytosis.

P. P. FOSTER AND M. R. HILL (*Archives of Dermatology and Syphilology*, April, 1940) note that a definite relationship exists between many attacks of *pruritus ani* and exacerbations of either seborrhœic eczema of the scalp, ear canals, eyelids, umbilicus and axillæ or of a dermatophytosis of the feet. They consider that there are many causes of *pruritus ani*, both local and constitutional, but feel that *pruritus ani* may be a toxic or an allergic manifestation of seborrhœic eczema or of dermatophytosis in the same way as the well-recognized dermatophytid of the hands is a manifestation of dermatophytosis. When *pruritus ani* arises as a secondary toxic or allergic reaction to a primary dermatophytosis or to a seborrhœic eczema, it is, by nature of the anatomy of the anus, in a position to become invaded by a variety of organisms, for example, yeast and pyogenic bacteria. The element of infection must be dealt with, as well as the primary focus causing the allergy. Other factors, such as scratching or abrasion by rough clothing, toilet paper, perspiration and anything which damages the lower layers of the epidermis and papillary bodies, must be considered. A summary of treatment is as follows: (i) Saline water on a soft cloth should be used instead of toilet paper. (ii) The part should be kept dry. (iii) Pathological conditions of the anus must be removed. (iv) Therapy should be continued for several months after the symptoms have subsided. (v) Constant attention must be paid to seborrhœic eczema and dermatophytosis of the feet. When these conditions recur, they should be actively treated.

The Treatment of Gravitational Ulcers.

F. F. HELLIER (*The Urologic and Cutaneous Review*, May, 1940) holds that the intractable nature of gravitational ulcers is due to actual or latent œdema. Unless this œdema is treated, all efforts to heal the ulcer usually prove fruitless. Weight should be reduced by dieting and exercises. Thyreoid gland is of benefit, not only for its weight-reducing effects, but also for its effect on the peripheral circulation. Anæmia should be treated by large doses of iron. In regard to local treatment, it is important to remove the œdema, the simplest method being to put the patient to bed with the foot of the bed raised and with a bland dressing, such as *Oleum Morrhuæ* 25 parts to *Unguentum Acidi Borici* 100 parts, applied to the ulcer. If this is impossible, the author advocates the use of "Elastoplast" bandages, which must be firmly and properly applied, the following points being observed: (i) Two side-pieces of "Elastoplast" are applied unstretched from just below the malleoli to just below the knee. (ii) The leg must be bandaged firmly, a start being made well down on the foot at the base of the toes and the bandage going well back to the heel. The bandage is applied firmly enough for the patient to complain of the tightness for an hour or so, and it is left on for a fortnight. If the patient is sensitive to "Elastoplast" the author uses "Viscopaste" bandages, bandages impregnated

with material very like Unna's zinc gelatine. A "Sorbo" rubber pad, with the external surface of the edges bevelled and considerably overlapping the ulcer, is a very useful addition to the treatment. The author applies the "Elastoplast" bandage to legs showing marked eczematization, provided there is no weeping. In the treatment of patients showing chemical sensitivity to "Elastoplast" he suggests that the bandage should be applied sticky side out or that it should be covered with gauze or a silk stocking before the "Elastoplast" is applied. When ulcers are very painful he dusts them with powdered aspirin before applying the bandage. Varicose veins, if present, are treated by injection of sodium morrhuate.

UROLOGY.

Variations in Form of the Renal Cavities.

B. FEY (*Journal d'Urologie*, September, 1939), who, with his master, Félix Legueu, has solved many of the mysteries of dynamism in the urinary tract, confesses that even after all his pyelographic and pyeloscopic experience the active cause of some varieties of hydronephrosis remains obscure. In particular cases, characterized by early dilatation and pain, the cause may be mechanical, congenital, neuro-muscular, endocrine or sympathetic; but it is impossible to decide which. At the renal level the orderly progression of the urine, like the same phenomenon lower down at the bladder outlet, depends on a harmoniously combined action of forces which are apparently antagonistic, that is, upon muscular synergism. As an analogy, the author quotes a simple instance of muscular physiology in the so-called voluntary sphere—for example, extension of the forearm on the arm. Isolated contraction of the triceps would cause a brusque, imprecise, ataxic movement. It is necessary that the biceps, its so-called antagonist, should also contract in such a way that it can then relax its contraction in order to allow, little by little, predominance of the movement of extension. It is from this synergism that the harmony and precision of our bodily movements come. It takes very little to upset such a fine and delicate mechanism, especially when such a small and fine structure as the outlet from the renal pelvis is concerned. The surgeon should discard the old and purely mechanical outlook on the physiology of the renal pelvis and calyces and try, in every possible case, conservative measures which may give the plain muscle a chance to recover its full physiological function. Much more physiological knowledge is required, but at present operations of denervation, drainage of the pelvis and removal of obvious obstacles to outflow will often secure vast improvement.

Vesical Neck Obstruction in the Female.

P. THÉVENNARD (*Journal d'Urologie*, October, 1939) draws attention to the occurrence of so-called "prostatism" in the female. He recalls the work of Caulk on this disease, and describes personal cases to illustrate his article. The outstanding symptom is progressively increasing difficulty in micturition. Later, proper vesical emptying is not achieved and frequent desire to empty out only small amounts of urine supervenes. The residual urine mounts in quantity, with a suprapubic sensation of weight, and finally complete retention of urine appears as an acute emergency. Later developments are renal back pressure even with bilateral hydronephrosis and uræmia; also incontinence of overflow. The appearance of cystitis complicates and modifies the above symptomatology. In some cases endoscopy reveals a definite obstruction at the internal meatus, which may be carefully removed by endoscopic electric resection; but in many instances nothing of importance can be determined by cysto-urethroscopy. In the differential diagnosis the surgeon must be careful not to overlook the possibility of *tabes dorsalis* or other diseases of the spinal cord which may cause atony of the bladder, thus simulating obstruction. Locally it is possible to confuse the effects of stricture of the urethra or cystocele with those of obstruction due to disease of the bladder neck itself. In treatment the operation of choice is that of endoscopic resection through the urethra. When the result is poor by this method the surgeon should use the suprapubic operation of ablation of the bladder neck devised by Marion. The dangers of endoscopic operation in the female are: (i) permanent incontinence and (ii) vesico-vaginal fistula. A special loop electrode has been designed by Hendrickson for use with the McCarthy resectoscope, by which much smaller pieces of tissue are removed by each excursion of the loop. With care and skill the above accidents may easily be avoided. Instead of being confined to the floor of the internal meatus, the resection should be circumferential, and in any case of doubt the resection may be carried out in two sessions.

Bactericidal Effect of Sulphathiazol and Sulphamethylthiazol.

H. F. HELMHOLZ (*Proceedings of the Staff Meetings of the Mayo Clinic*, February 21, 1940) compares the effect of sulphanilamide with the new sulphonamide derivatives on *Streptococcus fæcalis* and *Staphylococcus aureus*. The former is, for practical purposes, of no avail against these organisms. The author found in all his experiments *in vitro* that both sulphathiazol and sulphamethylthiazol have a very decided bactericidal action against both types of infection and that this action is much greater than that on the Gram-negative organisms. The concentration required is 150 milligrammes per 100 cubic centimetres of urine. Sulphamethylthiazol is the more effective. He also found that five strains of *Proteus ammoniæ* were readily destroyed by both drugs at a pH of 5·5.

T. I. POOL AND E. N. COOK (*ibidem*) record their results with sulphathiazol in the treatment of fifteen and with sulphamethylthiazol in the treatment of thirty-five patients suffering from urinary infection. In 65% the urine became and remained sterile, a figure which corresponds to the results obtained with other drugs. They state, however, that in the cases which failed to respond, the concentration of the drug was less than 150 milligrammes per cubic centimetre of urine, whereas in the successful cases the concentration exceeded this figure. The authors therefore suggest that the dosage used by them (one gramme four times a day) should be increased. Special mention is made of *Staphylococcus aureus*, which was found in the urine in five cases; in all the urine was rendered sterile. Of seven patients in whose urine *Streptococcus fæcalis* was detected, three were cured. The remainder apparently had an inadequate dose. These two organisms are notoriously difficult to eradicate from the urinary tract, even with adequate doses of other sulphonamide compounds. It is also concluded that the new derivatives are less toxic than either sulphanilamide or sulphapyridine.

Extraction of Large Renal Calculi.

L. SURRACO (*Journal d'Urologie*, September, 1939) describes a method of "extended pyelostomy" which enables the surgeon to avoid the more serious operation of nephrotomy in attempting removal of large or branching renal calculi. A semicircular incision is made in the renal capsule covering the posterior surface of the kidney. A flap of capsule, comprising about one-half of the posterior portion of the membrane, is turned down over the renal pelvis. It carries down with it, and covers over, the retropelvic artery and vein. The surgeon's finger is inserted between the posterior lip of the renal sinus and the capsular flap, and the extension of the capsule on the inner surface of the renal sinus is separated from the latter by a continuation of the blunt dissection. The flap of capsule is then divided with scissors right into the depth of the renal sinus. The retropelvic veins may need clamping during this manœuvre, but the corresponding artery will usually escape injury. Elevation of the posterior lip of the renal sinus by means of a suitable retractor will now afford the operator an excellent view of the whole of the renal pelvis, with the three major calyces (infundibula) running off it, and incisions can be made where desired. The necks of the infundibula can be dilated if necessary, and suitable forceps or even the finger tip can be passed into the calyces. Most kinds of calculi can be removed by this operation, which can, however, be combined with very limited nephrotomy incisions over individual calyces if necessary.

Renal Position of an Accessory Suprarenal Capsule.

A. ALEXANDRE (*Urologia*, June, 1939) reports the discovery of a small but complete suprarenal capsule lying on the surface of a kidney near the upper pole. The condition, which is rare, was discovered during a nephrolithotomy. A fairly large accessory polar artery entering the upper pole had to be divided, with consequent appearance of an infarct on the surface. In the centre of the latter area there was clearly visible under the renal capsule an oval yellowish area. Since it was impossible to decide whether he was dealing with an accessory suprarenal body or an actual small Grawitz tumour, the author made a small cruciform resection and completely removed the suspected area. It was discovered that the tissue was a suprarenal gland in miniature, with its own fine, but complete, fibrous capsule. There was no connexion between its vascular system and that of the renal cortex, but at one point an artery entered the miniature gland. This artery without doubt came from the accessory renal vessel previously divided. The theory of Pepere is recalled, which assigns to blood vessels a fundamental role in the transportation of aberrant suprarenal rests, the implications concerning the origin of hypernephroma being included in the theory.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

VIII.

EPILEPSY.

"He hath the falling sickness."

EVERY practitioner knows that many sufferers from epilepsy are not insane and that they are able to hold positions, sometimes high and responsible positions, and to perform their duties with only occasional interruptions from their malady. It is therefore necessary for a patient to have some mental disturbance in addition to epileptic fits to come under the diagnosis of insanity with epilepsy. The most usual of these conditions are: (*a*) congenital mental deficiency, (*b*) epileptic hallucinations, (*c*) epileptic confusion, (*d*) epileptic amnesia, (*e*) epileptic dementia. Of course, two or more of these conditions may be associated in one case and a patient may have hallucinations, interspersed with periods of confusion, and after a longer or shorter period become mentally enfeebled.

Conditions Associated with Epilepsy.

Mental Deficiency.

A child who becomes subject to fits in infancy is always liable to suffer from mental deficiency. Sometimes the same pathological condition, as birth trauma or hydrocephalus, will cause both the deficiency and the epilepsy; sometimes the fits arise after infantile convulsions of various causation, and the brain, having, as it were, got into the habit of having fits, continues the procedure after the original cause has disappeared.

Mental deficiency combined with epilepsy has no special significance except that the child is particularly difficult to train. After months of hard work on the part of the teacher, just as the child appears to be making progress, a series of epileptic fits will occur and the child forgets all he has been taught and the teacher has to start again from zero.

Hallucinations.

Hallucinations, both visual and auditory, are frequently associated with epilepsy. These hallucinations may arise suddenly and are often of a religious nature. They give the impression that they are a regression to infancy and the patient sees visions resembling the picture books of his early childhood days. The Roman Catholic sees visions of the saints, the Protestant sees God on a throne surrounded by angels, and one patient of mine, who was a Jew, saw the Ark of the Covenant. These visions are particularly vivid and are often accompanied by auditory hallucinations of an appropriate character and the patient is instructed to save the world or to preach the gospel. Possibly these hallucinations account for the ancient name of *"morbus sacer"*, and it is interesting to note that both Swedenborg and Mohammed were epileptics.

Confusion.

Confusion may occur after fits. The patient is restless, wanders aimlessly, is unable to state where he is or to recognize people. He is sometimes resistive, is unable to grasp what is said to him, and is intractable and difficult to manage. Sometimes these states of confusion pass into what is called epileptic furor, when the patient becomes very violent, is liable to attack anyone and may commit murder. The patient is always amnesic after these attacks of confusion, and quite honestly says that he cannot remember anything about them. In my opinion the violent epileptic is the most dangerous of all insane persons. There is a popular belief that the madman has superhuman strength. This, of course, is untrue: the madman has strength according to his size and physique, but the epileptic does not appear to care whether he hurts himself, while the normal man in a fight is usually careful not to hurt himself, however much he desires to hurt others. This lack of self-consideration appears to make the epileptic stronger and more dangerous.

This condition of confusion or furor does not usually last more than two or three days, and sometimes passes off in a few hours, when the patient becomes quieter, slowly returns to a reasonable frame of mind and asks where he is and what he has been doing. Sometimes he is very distressed when told how violently he has behaved, and sometimes refuses to believe it. When once a patient has had an attack of confusion or furor, this mental condition may recur after any fit; but, on the other hand, the patient may have periodical fits for the rest of his life and never again behave violently.

Amnesia.

Epileptic amnesia may occur after the confused state or may occur without it. The patient may wander about, sometimes for days, and during this time may perform perfectly normal actions, conduct his business, travel by train or visit friends and no one realizes that he is mentally abnormal. He may then return to his normal condition, but with loss of memory for the time he was in this condition of epileptic trance or fugue, and he cannot be held responsible for his actions during this period.

Dementia.

Long-standing epilepsy lasting over a number of years frequently leads to the loss of mental faculties. The patient gradually loses his interest in the outside world, in his business, his friends and relatives, and finally in himself. He becomes dirty and untidy, is unable to care for himself, and is finally reduced to a vegetative existence.

Diagnosis.

Any of these mental conditions may arise after *grand mal* or *petit mal* fits. Jacksonian fits rarely cause insanity. If the history of previous epileptic fits can be obtained the diagnosis is easy; if not, it is extremely difficult. Epilepsy may be suspected from the religious character of the hallucinations and from the suddenness of the onset and the violence of the outbreak; but until the history can be obtained the diagnosis often has to be left in doubt. Confusion from other causes, say cerebral tumour, must be excluded, and sometimes the patient's condition resembles acute mania or hysteria.

Treatment.

"Luminal", half a grain four times a day, is the regular treatment. "Prominal" has been recommended. I find that some patients are much better on "Luminal sodium" solution, half a grain four times a day, than on solid tablets. Bromides can be added to the "Luminal sodium" if desired; but bromides, if administered for long, appear to increase the mental enfeeblement. "Bromurol" or "Dilantin" can be tried. If any of these drugs are given, they must be reduced very slowly; after the patient has been without any fits for six months the dose could be reduced by half a grain a day, and so on, for each six months. If the "Luminal" is suddenly stopped, *status epilepticus*, in which the patient has a succession of fits, say twenty in half an hour, may ensue and the patient dies. For this condition of *status epilepticus* intramuscular injections of "Luminal" solution, one cubic centimetre of 20% solution (Bayer Products) should be given every half-hour till the fits cease.

For epileptics who become irritable before or after the fits, the great principle of treatment is to leave them alone as much as possible. The patient should be put to bed in a room, the door left open so that he can be observed without being disturbed, and food placed on a table by his bed and left for him. His medicine must be given, but the friends must not come in every hour to ask whether he wants anything. The best intentions of the relatives serve only to irritate the patient. Whilst he is in this irritable state the food must be in soft or liquid form so that there is no chance of his choking. The bottle containing the "Luminal" must on no account be left in his room for him to help himself.

A good nurse, or sometimes a relative, is able to tell when the patient is what is called "fitty" and knows that the patient will have a fit in a few hours. Patients who are liable to become irritable or have any of the above-mentioned mental symptoms, should be put to bed in anticipation of the fit.

All epileptics must abstain from alcohol.

Sometimes the physician is called in to see a patient who is already in a state of epileptic furor, and it is obvious that something must be done at once on account of the violence of the patient and the danger to his wife and children. In this case a hypodermic injection of

Hyoscine hydrobromide, gr. $^1/_{50}$
Morphine sulphate, gr. $^1/_6$
Atropine sulphate, gr. $^1/_{100}$

should be given at once and the patient restrained until it takes effect, which should be within thirty minutes.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on May 15, 1940, at the Royal Melbourne Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Part of this report appeared in the issue of August 10, 1940.

Lobar Pneumonia and Pericardial Effusion Treated with "M & B 693".

DR. DOUGLAS THOMAS showed a male patient, aged twenty years, who had been admitted to hospital on February 14, 1940, complaining of stabbing pain in the left side of the chest, worse on coughing or taking a deep breath. He reported having had some night sweats prior to his admission to hospital.

Examination revealed that the patient's temperature was 103·4° F.; his pulse rate was 104 and his respirations numbered 40 per minute; the systolic blood pressure was 110 and the diastolic pressure 70 millimetres of mercury. The heart sounds were normal. The apex beat was in the fifth left intercostal space, three and a half inches from the mid-line. The patient was given "M & B 693", two tablets every four hours. On the following morning he had faint signs of consolidation at the base of the left lung.

On February 20, 1940, the signs at the base of the left lung were unaltered; but pronounced pericardial friction was now audible. The heart sounds were rather fainter, but were easily heard. Some crepitations were detected on February 25, 1940, over the middle lobe of the right lung; a friction rub was audible over that area on February 26, 1940. A rash developed and the sulphapyridine treatment was temporarily discontinued. The heart sounds were now fainter, although the friction rub could still be heard. An X-ray examination disclosed a moderately large pericardial effusion.

On February 28, 1940, a needle was inserted into the pericardial sac and four cubic centimetres of straw-coloured fluid were removed. This proved to be sterile. An X-ray examination on March 7, 1940, showed the heart shadow to be still enlarged and a little fluid was present at the base of the left lung. The patient was very much better, although his temperature was still somewhat elevated. On March 16, 1940, a needle was inserted into the base of the left pleural sac and a little straw-coloured fluid was removed. On March 28, 1940, he still had a slight elevation of temperature. An X-ray examination then showed the bases of the lungs to be normal, but some pericardial effusion still existed. From then on he made an uneventful convalescence. When he was discharged from hospital on April 20, 1940, his cardiac outline was normal, according to X-ray examination.

Pulmonary Asbestosis and Pernicious Anæmia.

Dr. Thomas then showed a male patient, aged sixty-two years, who had been admitted to the wards complaining of general weakness, dyspnœa, vague pains in the chest and great loss of weight. He had been a worker in the asbestos trade for eighteen years. The work was dusty and involved the handling of asbestos in a dry state.

On examination he was found to have a normal temperature. The systolic blood pressure was 140 and the diastolic pressure 90 millimetres of mercury. No definite abnormality was detected in the heart. Examination of the lungs revealed signs of patchy consolidation and emphysema. The abdomen was normal. Examination of the central nervous system disclosed pronounced wasting of the small muscles of both hands. General muscular hypotonia was present. There was some loss of finer sensation in both hands.

X-ray examination disclosed a fine generalized fibrosis throughout both lungs. No evidence of tuberculosis was apparent. Examination of the sputum failed to reveal tubercle bacilli; but asbestos bodies, of the typical drumstick pattern, were found. Examination of the blood revealed a macrocytic anæmia; the erythrocytes numbered 2,000,000 per cubic millimetre, the hæmoglobin value was 50% and the colour index was 1·25; the leucocytes numbered 6,800 per cubic millimetre. A slight shift to the left was revealed by the Arneth count. He was given "Campolon" and responded very well. Large quantities of vitamin B_1 were given at the same time. His hæmoglobin value rapidly rose to 84% and the erythrocyte count to 4,700,000 per cubic millimetre. At the time of the meeting sensation was almost normal in the hands and the muscles were filling in. Apart from superficial keratitis, probably caused by silica particles, no other important abnormality had been discovered. A minor degree of osteoarthritic spondylitis was present in the cervical portion of the spine; but this was not considered to be related to the wasting of the muscles in the hands.

Psoriasis.

DR. R. R. WETTENHALL showed a number of patients suffering from psoriasis. The first was a female patient, aged eighteen years, who had had recurrent attacks involving the elbows, knees, back, scalp and the area behind the ears; the disease was of doubtful duration. On May 11, 1939, she had been treated at the Royal Melbourne Hospital with *Haustus Sodii Salicylatis, Unguentum Acidi Salicylatis, Olei Cadini* and *Unguentum Hydrargyri Ammoniati*. On May 13, 1940, a few affected areas were still present. Dr. Wettenhall drew attention to three points of interest: (i) the patient's comparative freedom from attacks, due to appropriate treatment; (ii) the necessity for entirely clearing up an attack of psoriasis, as remnants, no matter how small, predisposed to an earlier occurrence of the next attack; (iii) the patient's good response to treatment.

The next patient shown by Dr. Wettenhall was a female, aged forty-two years. She had had indigestion for years, and investigation had revealed no abnormality. Five weeks prior to the meeting she had a sore throat with enlarged cervical glands. A scaly rash had been present on the left elbow for years. Two weeks prior to the meeting slightly irritable scaly spots had appeared on both arms and forearms and on the right leg.

On examination the patient was seen to have scaly erythematous macules, mainly affecting the flexor surfaces of both forearms. There was a silvery, scaly, erythematous plaque on the point of the left elbow. The tonsils were slightly enlarged and congested. A diagnosis of guttate psoriasis was made.

Dr. Wettenhall said that the case illustrated the probable influence of sepsis in causing an acute psoriatic eruption when there was a psoriatic tendency. The old psoriatic plaque on the left elbow was typical, as psoriasis had a predilection for the extensor surfaces and especially for the pressure points, such as the elbows and knees. The recent sore throat and the visible inflammation of the tonsils were probably the cause of the patient's acute eruption.

Dr. Wettenhall's third patient was a male, aged forty-eight years, suffering from psoriasis associated with superficial basal-celled epithelioma. He had since childhood suffered from psoriasis, which at times had been practically universal. He had had persistent reddish-coloured lesions in the region of the right shoulder and interscapular area for about twenty-three years. Examination revealed circumscribed infiltrated plaques of erythema with slightly raised borders in this area. A diagnosis of superficial basal-celled epithelioma was made. Dr. Wettenhall said that the condition had been kept under control by means of intensive doses of X-ray and radium therapy to the advancing nodular borders.

Dr. Wettenhall next showed a female patient, aged sixty-eight years, who had suffered from psoriasis for many years. She had had various forms of treatment at different times. Multiple persistent red lesions were present on the abdomen and chest. Examination revealed circumscribed infiltrated plaques of erythema with beaded edges. A diagnosis of psoriasis associated with superficial basal-celled epithelioma was made. Intensive treatment with X rays and radium was given to the nodular edges.

The next patient shown by Dr. Wettenhall was a male, aged fifty-three years, who had had areas of psoriasis on the elbows and knees for many years. He had had an acute attack four years earlier, for which he had been treated at the Royal Melbourne Hospital. The attack from which he was now suffering had begun fourteen days earlier; the eruption had rapidly spread over the arms, legs, trunk, scalp, face and jaw. The patient had not felt well lately. Dr. Wettenhall remarked that an acute attack frequently occurred in neurotic and poorly nourished persons. Psoriasis involving the face was rare; but the patient spent his time chiefly indoors and was probably not much exposed to light. In this case the attack coincided with the patient's feeling "below par". He was given soothing treatment, which was essential in acute cases; the introduction of stimulating treatment might easily lead to exfoliative dermatitis.

Dr. Wettenhall then showed a female patient, aged sixteen years, who had had numerous plaques, chiefly on the arms and legs, since the age of five years. The eruption had begun after an illness which suggested sensitization from sepsis; that was not infrequently noted. The patient had had no serious illnesses, and that also was usual. As far as was known, no other members of the family were affected. Dr. Wettenhall pointed out that ulceration was present; that was very rare and was suggestive of artefact.

Another patient shown by Dr. Wettenhall was a male, aged thirty-nine years, who had an extensive eruption, especially over the back. The affected areas became very hot and itchy at times. The patient worked underground, and his condition was suggestive of lack of daylight. The lesion resembled secondary syphilis, and a Wassermann test was performed. No infiltration was present and there was no abnormal adenitis. X-ray therapy had been given to the left arm for comparison with the right arm, which was untreated; the treated area was obviously improved.

Dr. Wettenhall finally showed a female patient, aged sixty-two years, whose elbows had been affected with psoriasis for twenty years. Her knees were also affected. She had an attack yearly, of variable severity. She was treated with salicylic acid, camphor, oil of cade and *Unguentum Hydrargyri Ammoniati*. X rays could be used. The chief point of interest was the typical situation.

Dr. Wettenhall discussed the differential diagnosis and treatment of psoriasis. He said that the differential diagnosis was from eczema, seborrhœic dermatitis, syphilis, *pityriasis rosea*, *lichen planus* and ringworm of the scalp or body. General treatment included the use of a diet of poor protein value, exposure of the patient to light, the avoidance of heat and the removal of nervous strain. The patient should go swimming. Internal treatment consisted in the administration of thyreoid extract, arsenic, potassium iodide and salicylate of soda. By way of local treatment, "Vaseline" could be applied, alkaline baths could be given, "Chrysarobin" and "Anthralin" could be used, and X-ray therapy could be applied.

Gall-Stones.

DR. G. R. A. SYME showed a married woman, aged fifty years, who had suffered from intestinal obstruction by a gall-stone. Some eighteen months earlier she had had an operation on the abdomen for perforation of a diverticulum. Eighteen hours before her admission to hospital on the present occasion she had had an attack of colic in the epigastrium, with some vomiting. Pain and vomiting had continued with slight intermission until the patient was vomiting dark foul material; the bowels had not been opened and the temperature was 99° F. There was no tenderness or distension, and after two successful enemata the vomiting and pain ceased. As, however, some dilatation and gaseous distension of the small bowel were visualized radiographically, Dr. Syme had operated and removed a large gall-stone from the ileum.

Dr. Syme also showed a married woman, aged forty years, who for six months had had attacks of pain in the right side of the hypochondrium, sometimes passing through to the back and sometimes radiating down to the right groin. Three months before her admission to hospital a severe attack of colic was associated with vomiting and sweating. She had had considerable flatulence and intolerance of fatty food, but had not been noticeably jaundiced. The urine was strong smelling and was passed rather frequently; it contained a few pus cells and motile bacilli. On occasions when the patient was examined Murphy's sign was elicited, but the kidneys were not palpable. The condition was regarded as due to gall-stones until on radiological investigation it was found that in addition to gall-stones a renal calculus was present. At operation cholecystectomy and pelvic lithotomy were performed through Kocher's incision, and the patient made an uneventful recovery.

Lateral Aberrant Thyreoid Tumour.

Dr. Syme also showed a married woman, aged thirty-seven years, from whom he had removed aberrant thyreoid tissue, microscopic sections of which he showed. For three months prior to operation the patient had noticed a lump on the left side of her neck, which was slowly increasing in size. It was found that she had a firm rounded tumour the size of a pigeon's egg under the sterno-mastoid muscle, approximately at the level of the angle of the jaw. Above it and below it were several discrete but less readily palpable tumours, which felt like enlarged lymph glands. One small superficial gland was excised for biopsy, and in it papillary epithelium was found; this suggested that the tumour was one of the lateral aberrant thyreoid group. The thyreoid gland itself was not definitely enlarged. Dr. Syme said that at the operation a long chain of semi-solid, semi-cystic swellings was dissected out from beneath the sterno-mastoid muscle; the swellings were not adherent and some were obviously lymphatic glands. Microscopic examination revealed that they were in part composed of papillary adeno-carcinomatous tissue, some of which had the appearance characteristic of thyreoid glandular material. Macroscopically, some of the swellings were seen to be simple cysts; but others contained blood or papillary tissue. Dr. Syme remarked that the tumours were usually only locally malignant, and if they were completely extirpated cures had been reported, though there was a tendency to recurrence even after ten years. They were not regarded as radio-sensitive and usually were associated with enlargement of the thyreoid gland.

(To be continued.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 153, of August 8, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Francis John Moss, M.B., B.S., John Courtney Abbot Dent, M.B., B.S., Charles Alexander Frew, M.B., Ch.M., Frank Vivian Munro, M.B., M.S., Frederick George Steele, M.B., B.S. (15th July, 1940) and Thomas Hewson Donnelly, M.B., B.S. (16th July, 1940).

Flight Lieutenant D. T. Shortridge is transferred from the Reserve to the Active List with effect from 15th July, 1940.

The notification appearing in the *Commonwealth of Australia Gazette* dated 4th July, 1940, concerning Frank Vivian Munro, M.B., M.S., is cancelled.

The notification appearing in the *Commonwealth of Australia Gazette* dated 18th July, 1940, concerning Stafford Howie Marsh, M.B., B.S., is cancelled.

Reserve: Medical Branch.

James Murray Moyes, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 22nd July, 1940.—(Ex. Min. No. 49—Approved 7th August, 1940.)

Correspondence.

THE REMOVAL OF INSECTS FROM THE EXTERNAL EAR.

SIR: While acting as resident medical officer to the Seventh Field Brigade I was invited by the battery commander of one of the batteries to attend a night exercise some miles from West Maitland. This night occupation of a battery position is carried out in absolute silence and without lights. In the middle of the procedure one of the gunners rushed to me in great distress, complaining that something had flown into his ear and was driving him mad. He was clearly in great distress, and I had no means of giving first aid at hand. However, fortunately I remembered I had a small fountain pen pocket torch in my pocket and, arguing that insects usually flew towards light, applied this to the external auditory meatus for some fifty seconds, at the end of which time, to my satisfaction and the patient's intense relief, out crawled the offending insect and flew away.

The above procedure is very simple and elementary, and yet to my knowledge has never been suggested as a means of treating the above conditions.

Yours, etc.,

IAN F. VICKERY,
(Capt.) A.A.M.C.

2/4th Field Ambulance,
Ingleburn,
August 3, 1940.

THE TREATMENT OF CONTUSIONS AND WOUNDS.

SIR: Owing to the need for economy in cotton goods, I would like to offer to members of our profession, especially those associated with hospital and ambulance administration, a suggestion whereby they could not only effect considerable economy, but also improve their service.

I refer to the present method of treatment of contusions and wounds. The tendency amongst many people is to advise that the wound be unbandaged and a hot bath or

some other form of treatment given two or three times daily. It is doubtful whether disturbing the dressing does the least good, and, in fact, in some cases it probably does harm. All that is needed in the average case is for the wound to be dressed with an antiseptic dressing and kept securely covered for several days, unless abnormal pain or signs of spreading inflammation warrant an inspection. Rest should be the most important part of the treatment. This brings us to the purpose of this letter, which is a suggestion for economy in bandages and more secure bandaging. The "Gauzetex" type of bandage offers the best means of obtaining this result, but it is imported and costly. As a substitute I would recommend that every first-aid post should have a bot of bandage paste and a brush to apply it. A few turns of a bandage reasonably well stuck with paste will usually be retained far more effectively than a full bandage applied in the orthodox manner.

The enormous saving that will be effected must be obvious to all of us, who frequently see a whole bandage length used to cover some simple injury. We have experimented with pastes and have found a suitable formula is: Davis gelatin liquid glue into which sufficient zinc oxide powder has been added to give it a white colour; a few drops of phenol should also be added for antiseptic purposes.

Yours, etc.,

L. J. JARVIS NYE.

Wickham Terrace,
Brisbane,
August 2, 1940.

Analytical Department.

"CHEFOL."

"CHEFOL" is a brand of peanut oil. It is a product of Nut Foods, Proprietary, Limited, of Marrickville, Sydney. It is represented by the manufacturers to be a suitable substitute for olive oil for either internal or external use. A sample was purchased on the open market and submitted to analysis. Our analyst's report is as follows:

Specific gravity (15·5°/15·5° C.)	0·918
Refractive index at 40° C.	1·4630
Acid value	0·04
Saponification value	189·3
Iodine value	92·2

B.P. (1932) test for absence of other vegetable oils: sample passes test.

B.P. (1932) test for absence of cotton-seed oil and sesame oil: sample passes test.

Solubility in alcohol: slight.

The sample is miscible with ether, chloroform, and light petroleum (boiling point 50°-60° C.).

The sample conforms in all respects with the B.P. (1932) specification for arachis oil.

Peanut oil closely resembles olive oil, and its use as a substitute for olive oil is permissible. To medical practitioners who wish to prescribe peanut oil "Chefol" can be confidently recommended.

Obituary.

EDWARD SYDNEY LITTLEJOHN.

WE are indebted to Dr. J. I. C. Cosh for the following appreciation of the late Dr. Edward Sydney Littlejohn.

The earliest recollection I have of Dr. Littlejohn goes back to school days at the Sydney Grammar School, when he was known as Littlejohn I. Later he went to the University of Sydney and took his degree of B.A. For his medical education he went to Edinburgh and in due course graduated in medicine. Later on he had the degree of M.D. conferred upon him by the same university. Some years were then spent in the service of the cable station at Malta.

On returning to Sydney he entered general practice at Darlinghurst; moving soon to Croydon, he succeeded to the practice of Dr. Spiers Kirkland. It was here that we again met. The same reserved manner which he had at school had become even more marked. On this account he was a man difficult to know. When honorary secretary of the Western Suburbs Medical Association I tried, without success, to get him to come to our meetings and give us the benefit of his large experience in diseases of children. At this time Dr. Littlejohn was assistant physician to the Children's Hospital, which had its out-patient department in a building in George Street. In due course he became honorary physician to the Royal Alexandra Hospital, of which, at the time of his retirement from the staff, he was senior physician. For many years he was visiting medical officer to the Thomas Walker Hospital for Convalescents and continued this work until his death.

His leisure time at home was spent largely in the seclusion of his well-filled library—another evidence of his retiring disposition. For outdoor recreation he found golf had its attractions; he was a member of the original Strathfield Club, which had its course near the Boulevarde. When the club amalgamated with the Concord Club he became a life member—I have been told that after the change he went right off his game. Dr. Littlejohn worked right up to a few weeks before his death. When he realized that the end was not far off he accepted his fate with calmness and with no regrets—he had lived an upright life, full of helpfulness to the many suffering children to attend whom it had been his great privilege.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Downing, John Martin, M.B., B.S., 1937 (Univ. Melbourne), Devon Hospital, Latrobe.

Black, Percy Webber, L.R.C.P.I. and L.M. (Ireland), 1911, L.R.C.S.I. and L.M. (Ireland), 1911, Kingston.

Stenhouse, Barbara Graham, M.B., B.S., 1938 (Univ. Melbourne), Bothwell.

VICTORIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as a duly qualified medical practitioner:

Dittmer, Felix Cyril Sigismund, M.B., B.S., 1930 (Univ. Sydney).

The following additional qualification has been registered:

Murphy, Gregory B. V. (M.D., 1938, Univ. Melbourne), M.R.C.P. (London), 1940.

Honours.

HIS Majesty the King has been pleased to appoint Colonel Wilfred Vickers, D.S.O., to be a Commander of the Grand Priory of the Order of the Hospital of Saint John of Jerusalem.

NOTICE.

THE next clinico-pathological conference arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology will be held at the Prince Henry Hospital, Little Bay, New South Wales, on Monday, August 26, 1940, at 4.30 o'clock p.m. The subjects will be "A Case of Hæmatemesis with Unusual Features" and "A Neurological Case for Discussion and Diagnosis". A cordial invitation to be present is extended to all medical practitioners.

Books Received.

"Collected Papers of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne: Volume X, Containing 42 Papers"; 1940. Melbourne: The Walter and Eliza Hall Institute of Research in Pathology and Medicine. Demy 4to.

"Notter and Firth's Hygiene", revised by L. C. Adam, M.B., B.S., and E. J. Boome, M.B., Ch.B., M.R.C.P., D.P.H., T.D.; Tenth Edition; 1940. London: Longmans, Green and Company. Demy 8vo, pp. 518, with illustrations. Price: 12s. 6d. net.

"The Diagnosis and Treatment of Diabetes", by W. W. Ingram, M.C., M.D., F.R.A.C.P.; Third Edition; 1940. Australia: Angus and Robertson Limited. Crown 8vo, pp. 159. Price: 6s. 6d. net.

"Bacillary Dysentery in Children at Batavia", by B. L. Van Bueren; 1940. Batavia: The Children's Department of the Medical College. Medium 8vo, pp. 208, with illustrations.

"The Neuroses and Psychoses of War", by A. Frank, M.D.; English Edition; 1940. Australia: Angus and Robertson Limited. Large crown 8vo, pp. 93. Price: 4s. 6d. net.

"Atoms in Action. The World of Creative Physics", by G. R. Harrison; 1940. London: George Allen and Unwin Limited. Demy 8vo, pp. 380. Price: 12s. 6d. net.

"The Diabetic A B C. A Practical Book for Patients and Nurses", by R. D. Lawrence, M.A., M.D., F.R.C.P.; Seventh Edition; 1940. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 71. Price: 3s. 6d. net.

"Practical Handbook of the Pathology of the Skin. An Introduction to the Histology, Pathology, Bacteriology and Mycology of the Skin, with Special Reference to Technique", by J. M. H. Macleod, M.A., M.D., F.R.C.P., and I. Muende, M.B., B.S., B.Sc.; Second Edition; 1940. London: H. K. Lewis. Royal 8vo, pp. 436, with illustrations, many of which are in colour. Price: 42s. net.

"Medical Research Council of the Privy Council. Special Report Series, No. 241: Dental Disease in the Island of Lewis; 1940. London: His Majesty's Stationery Office. Medium 8vo, pp. 63. Price: 1s. net.

"The Compleat Pediatrician: Practical, Diagnostic, Therapeutic and Preventive Pediatrics", by W. C. Davison, M.A., D.Sc., M.D.; Third Edition; 1940. Durham: Duke University Press. Royal 8vo, pp. 262. Price: $3.75 net.

"Surgical Anatomy", by G. Massie, M.B., M.S., F.R.C.S.; Fourth Edition; 1940. London: J. and A. Churchill Limited. Medium 8vo, pp. 480, with 158 illustrations. Price: 21s. net.

"Clinical Electrocardiography", by D. Scherf, M.D., and L. J. Boyd, M.D., F.A.C.P.; 1940. London: William Heinemann (Medical Books) Limited. Medium 8vo, pp. 374, with illustrations. Price: 25s. net.

"Second Addendum to the British Pharmacopœia, 1932"; published under the direction of the General Council of Medical Education and Registration of the United Kingdom; 1940. London: The General Medical Council. Medium 8vo, pp. 32.

"The Universe through Medicine", by J. E. R. McDonagh, F.R.C.S.; 1940. London: William Heinemann (Medical Books) Limited. Demy 4to, pp. 389, with illustrations. Price: 25s. net.

"Forensic Chemistry", by H. T. F. Rhodes, Dip.Inst.C.; 1940. London: Chapman and Hall Limited. Demy 8vo, pp. 222. Price: 12s. 6d. net.

Nominations and Elections.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Alexander, John Murray, M.B., B.S., 1937 (Univ. Sydney), 4, Tryon Avenue, Wollstonecraft.

Burgess, John Sanday, M.B., B.S., 1940 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Humphery, Frederick Thomas, M.B., B.S., 1940 (Univ. Sydney), Lewisham Hospital, Lewisham.

Read, George, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

Shea, Leonard Thomas, M.B., B.S., 1938 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Medical Appointments.

Dr. W. T. Dermer has been appointed Medical Officer of Health to the Youanmi Local Board of Health, according to the provisions of *The Health Act*, 1911-1937, of Western Australia.

* * *

The following have been appointed Members of the Medical Board of South Australia: Dr. L. W. Jeffries, Dr. E. A. Johnson, Dr. P. S. Messent, Dr. H. H. E. Russell, Dr. A. M. Cudmore.

* * *

The following temporary appointments to the honorary medical staff of the Royal Adelaide Hospital, Adelaide, have been made: Assistant Physician, Dr. M. W. Miller; Clinical Assistants to the Medical Section, Dr. F. G. T. Turner, Dr. E. B. H. Brotchie; Clinical Assistant to the Orthopædic Section, Dr. N. S. Gunning.

The following honorary appointments have been made at the Royal Alexandra Hospital for Children, Camperdown, Sydney: Surgeons, Dr. L. G. Tait, Dr. C. H. Wesley; Assistant Surgeons, Dr. A. F. Hobson, Dr. E. H. Goulston; Temporary Relieving Assistant Surgeons, Dr. G. M. B. Hales, Dr. B. Denning, Dr. M. Sofer-Schreiber, Dr. M. M. Kennedy; Temporary Relieving Assistant Physicians, Dr. M. E. Hynes, Dr. C. Warburton; Ear, Nose and Throat Surgeon, Dr. W. Ramsay Beavis; Temporary Relieving Assistant Ear, Nose and Throat Surgeon, Dr. M. L. Coutts.

Diary for the Month.

AUG. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 28.—Victorian Branch, B.M.A.: Council.
AUG. 29.—New South Wales Branch, B.M.A.: Branch.
AUG. 29.—South Australian Branch, B.M.A.: Branch.
AUG. 30.—Tasmanian Branch, B.M.A.: Council.
SEPT. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 3.—Federal Council, B.M.A.: Meeting at Sydney.
SEPT. 4.—Victorian Branch, B.M.A.: Branch.
SEPT. 4.—Western Australian Branch, B.M.A.: Council.
SEPT. 5.—South Australian Branch, B.M.A.: Council.
SEPT. 6.—Queensland Branch, B.M.A.: Branch (Jackson Lecture).

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associate Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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